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ROENTGENOLOGIC APPEARANCE AND PATHOLOGY OF INTRAPULMONARY LYMPHATIC SPREAD OF METASTATIC CANCER

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THE lymphatic spread of metastatic cancer in the lung presents a rather unusual roentgenologic appearance and is seen with sufficient frequency to justify greater interest than has been shown in the past. Its roentgen recognition is particularly important when the patient's early or presenting symptoms are referable to the chest only. It seems worth while, therefore, to report 10 cases which have been seen at the Massachusetts General Hospital in recent years. Seven of these cases were autopsied, while the remaining 3, who were known to have cancer, developed, while under observation, roentgenologic changes typical of lymphatic spread.

CASE REPORTS

CASE 1. (B. I. U-406494) The patient, female, aged fifty-four, developed a productive cough and pain in the region of the lower sternum five months before admission. During the last two months the sputum was occasionally blood streaked. There were no chills or fever. Despite periods of remission she became progressively worse, and three weeks before entry developed marked respiratory difficulty with asthmatic wheezing.

On admission the patient was slightly dyspneic, with a rapid pulse, and a few coarse râles could be heard over the left chest; otherwise the lungs were clear. Under a clinical diagnosis of chronic bronchiectasis and asthma the patient was bronchoscoped. The mucosa of the right main bronchus was friable and bled easily, but these changes were believed to be inflammatory in nature. Biopsy was attempted, but no tissue could be obtained. The patient became gradually worse, with marked dyspnea, and latterly cyanosis as the most prominent clinical feature of her illness. These symptoms were not alleviated by aminophyllin, adrenaline or morphine, though an oxygen tent gave some subjective relief. Death occurred suddenly on the eleventh hospital day.

Roentgenologic examination of the chest showed enlargement of both hilar shadows, particularly on the right side. There was a diffuse, string-like and beaded design of increased density radiating from the hilum toward the periphery on both sides, interspersed with fine nodular areas which lay partially within and partially without the trabeculated network of increased density. The changes were evenly distributed over both lungs, but were more marked in the bases than in the upper lung fields, and more in the central than in the peripheral portions. A small amount of fluid was seen in each pleural cavity. A later roentgenogram showed evidence of destruction of the lateral portion of the ninth right rib (Fig. 1).

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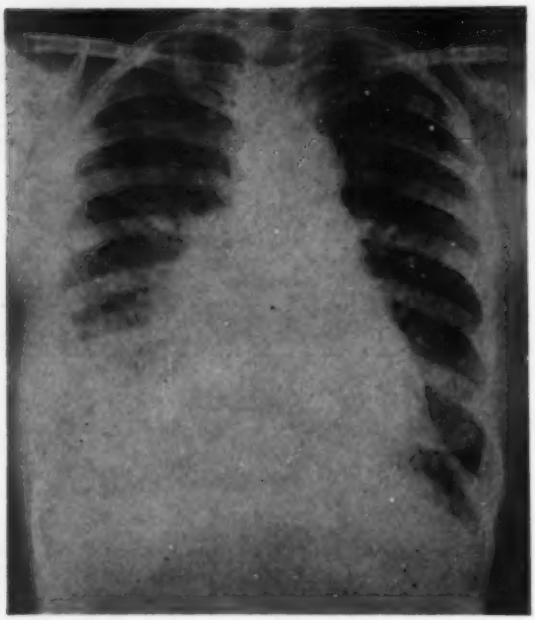

FIG. 1. Case 1.

The original diagnosis was bronchiectasis and fibrosis.

Anatomic Diagnosis. Carcinoma of the pancreas with metastases to lungs, bronchial and retroperitoneal lymph nodes; bronchiectasis.

At postmortem, the origin of the primary tumor was not immediately obvious, but the pancreas, which was of normal weight and size, contained scattered granular nodules, which averaged 1 cm. in diameter, and its normal lobular architecture had been destroyed. The left pleural cavity contained a liter of clear fluid and was free from adhesions. The right pleural cavity was obliterated by fibrous adhesions except for a 200 cc. collection of cloudy fluid above the diaphragm posteriorly. The lymphatics of the visceral pleura of each lung stood out as a prominent gray lacework and 1 to 2 mm. gray nodules had formed at the angles of this lacework. On section there was a moderate diffuse bronchiectasis with generalized congestion and edema. Throughout the parenchyma of each lung the lymphatics were also very prominent and beneath the mucosa of the bronchi were minute gray nodules. The bronchial and mediastinal lymph nodes had been invaded by tumor.

Microscopically the primary tumor was found to be an anaplastic carcinoma arising in the pancreas. The metastatic spread in the lungs was almost exclusively confined to the lymphatics of the pleura, septa, bronchial walls and perivascular lymph channels (Fig. 2). Tumor cells were lying in the peribronchial lymphatics as far out as the respiratory bronchioles. In addition, there were mild bronchiectatic changes and small areas of pneumonia and fibrosis. No blood vessel changes were detected, which could be considered unusual for the patient's age.

CASE II. (A. A. U-395930) Approximately one year before admission the patient, male, aged forty-six, developed anorexia and a sense of fullness in the epigastrium and began to lose weight. Four weeks before entry his symptoms became more severe and in addition he developed a cough, which was more marked at night, and productive of small amounts of white sputum without blood. One week before admission dyspnea and tachycardia were noted, and he perspired profusely without having chills or fever.

On admission, the patient was in moderate respiratory distress with a rate of 35, and moderate cyanosis was noted. The pulse was 100, the temperature 99° F. Examination of his chest showed a scattering of fine expiratory wheezes, a few coarse moist râles over both bases posteriorly, and a friction rub over the right base posteriorly. The abdomen was essentially normal. Biopsy of an enlarged cervical lymph node showed metastatic adenocarcinoma; the stomach was believed to be the probable seat of the primary tumor. The patient died twelve days after admission, at which time dyspnea, tachycardia, cyanosis and generalized pulmonary edema were the significant physical findings.

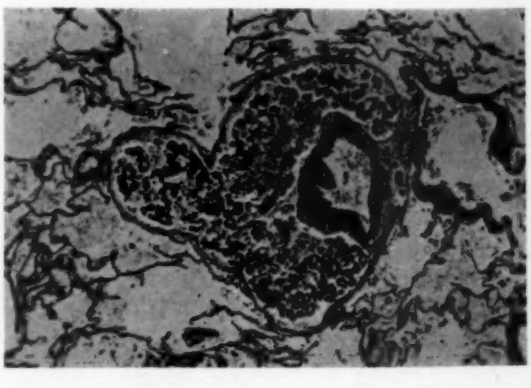

FIG. 2. Case 1. A dilated perivascular lymphatic filled with tumor cells.

Roentgenograms of the chest showed a rather marked, fine miliary, nodular and linear process involving both lungs in an uneven design of trabeculation which extended from enlarged hilar shadows toward the periphery and was most marked in the mid portions of the lungs, the apical and peripheral portions being least involved. Scattered within the miliary design there were a few larger areas of increased density, approximately 0.5 cm. in diameter (Fig. 3).

Anatomic Diagnosis. Carcinoma of the stomach with extension to the esophagus and metastases to the retroperitoneal, mediastinal, bronchial and cervical lymph nodes, kidney, lung and right adrenal.

At autopsy an annular tumor was found in the stomach arising in the region of the esophagogastric junction. Each pleural cavity contained approximately 200 cc. of cloudy fluid. The lungs were voluminous and the pleural surface on all aspects was studded with small gray nodules. The left apex was firm and airless, and here the pleura was covered with a fibrinous exudate. On section, the parenchyma was crepitant but somewhat edematous, and the consolidated area in the left apex had the appearance of pneumonia. In addition, many small gray nodules were scattered throughout the parenchyma and these seemed to be localized around the bronchi and blood vessels. There was an acute tracheobronchitis, and the mediastinal lymph nodes contained obvious metastatic tumor.

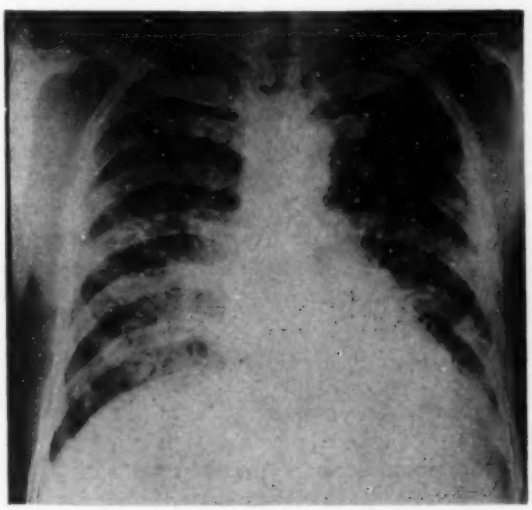

Fig. 3. Case II.

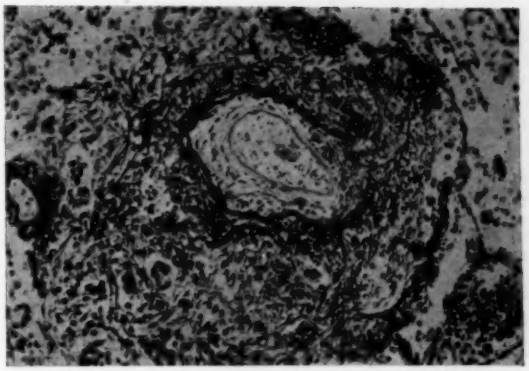

Fig. 4. Case II. A pulmonary artery showing concentric intimal thickening, a mild chronic inflammatory infiltration and tumor cells in the media and adventitia.

Microscopically, the primary tumor in the stomach proved to be a poorly differentiated adenocarcinoma with minimum gland formation. The lungs showed a patchy pneumonia and edema, but the most striking finding was the presence of poorly differentiated tumor cells lying almost exclusively within the dilated lymphatics of the bronchi, arteries and veins, and in some instances a sleeve of tumor cells had formed around these structures. Only minute clumps of tumor cells could be found in the parenchyma unrelated to the lymphatics. In addition, there were pronounced vascular changes, predominantly in the medium sized arteries and arterioles. The lumina of many contained tumor emboli in which the central cells were usually necrotic. Thrombotic material was often associated with these emboli thus leading to complete occlusion, and occasionally organization from the adjacent intima was well advanced. In a great many arteries the intima was thickened by a loose fibrous proliferation which was usually concentric, but occasionally took the form of a bud projecting into the lumen. Some of the smaller arteries were almost obliterated by this process. Tumor cells were sometimes incorporated in these segments of intimal thickening. The significance of this fact could not be determined; the matter will be discussed later. The media in many arteries contained small nests of tumor cells, while the adventitia with its tumor-filled lymphatics showed a dense fibrous thickening reminding one of the stromal reaction in the stomach wall. In most instances where tumor cells were present all layers of the artery showed a mild or severe infiltration of chronic inflammatory cells

(Fig. 4). Compression of arteries and veins by dilated tumor filled lymphatics was particularly noticeable.

CASE III. (M. M. U-101107) The patient, male, aged sixty, came to the hospital because of severe epigastric pain and vomiting which had been growing worse for the past four months. For five weeks the pain had been aggravated by deep breathing. Two days before entry he developed a cough, productive of thick yellow, frothy sputum, and dyspnea.

On examination, a few crackling râles were heard throughout the right lung, and the breath sounds were somewhat distant on the left. The pulse was always above 100 and at one point reached 160. The patient failed rapidly; his respiratory signs increased (râles and wheezes being present throughout the chest). Death occurred ten days after admission.

Roentgenologic examination showed a gross nodular involvement of the right lung and a network of irregular increased density which was evenly distributed over the entire lung, becoming more marked near the hilum. The left lung was less involved than the right, containing only a faint suggestion of a similar design. Re-examination one week later showed the changes in the left lung to have increased so that it presented a picture similar to that in the right lung—coarse nodularity connected by a network of linear markings. The process in the right lung had also become more extensive, and there was an area of homogeneous density in the central portion near the hilum which had not been present a week before. The process was more marked in the central and apical portions than in the lower lung fields (Fig. 5).

Anatomic Diagnosis. Carcinoma of the cardia of the stomach with perforation into the peritoneal cavity, with extension to the esophagus and pancreas, and with metastases to the liver,

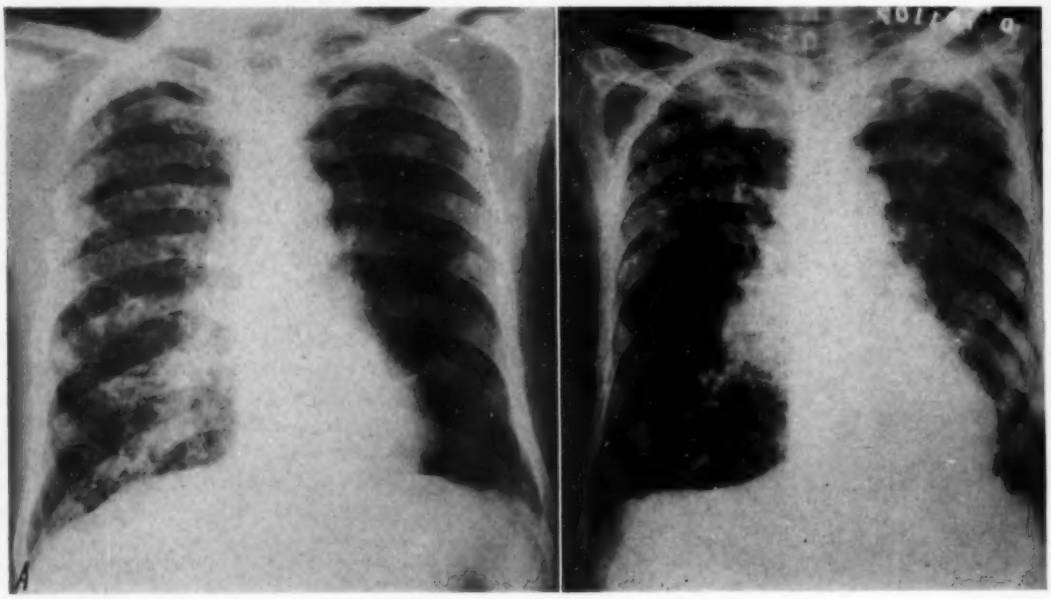

FIG. 5. Case III. *B* taken one week after *A*.

right adrenal, lungs, heart and regional and distal lymph nodes; acute fibrinous peritonitis.

Postmortem examination revealed a large ulcerating carcinoma, centered on the lesser curvature of the stomach. Perforation had occurred through the base of the tumor at two points. The pleural cavities each contained 300 cc. of clear fluid. The pleura of each lung was grossly normal except for many pinhead-sized subpleural nodules scattered over all surfaces. Some of these nodules, however, measured as much as 1 cm. in diameter. Section of the left lung showed a gross pneumonia in the lower half of the upper lobe with congestion and edema in the remaining parenchyma. The right lung was congested and edematous without obvious pneumonic consolidation. In each lung a cuff of firm gray tissue surrounded the cut ends of the

smaller bronchi. The trachea and opened bronchi seemed normal. The bronchial and mediastinal lymph nodes were enlarged to 2 cm. in diameter and contained gross tumor.

Microscopically, the appearance of the primary tumor in the stomach was one of extreme anaplasia and pleomorphism without gland formation. As anticipated from the gross appearance, the lungs showed innumerable collections of tumor cells throughout the parenchyma. These were situated primarily in the lymphatics of the pleura, interlobular septa, bronchial walls and around blood vessels. An occasional metastatic nodule was so large that this relationship was destroyed, and in a few fields groups of tumor cells lay free in alveolar spaces without causing tissue destruction. The lumina of a number of small and medium-sized arteries contained tumor cells which were often embedded in thrombotic material. This had led to occlusion of the smaller vessels, but in the larger arteries emboli were found clinging to the intima and here organization was well advanced, leaving a background of young fibroblasts in which tumor cells were sprinkled. The endothelium of the intima was resurfacing these buds of tissue. An occasional artery showed concentric tumor cell infiltration of all three coats with consequent marked intimal thickening leaving a central lumen (Fig. 6). In some fields alveolar capillaries had been flooded with tumor cells. Pneumonic consolidation was found in the lower half of the left upper lobe and there was a generalized but patchy edema.

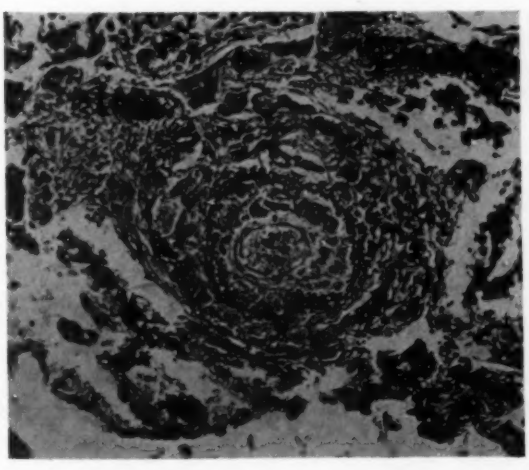

Fig. 6. Case III. A pulmonary artery with intimal thickening and tumor cell infiltration of all coats.

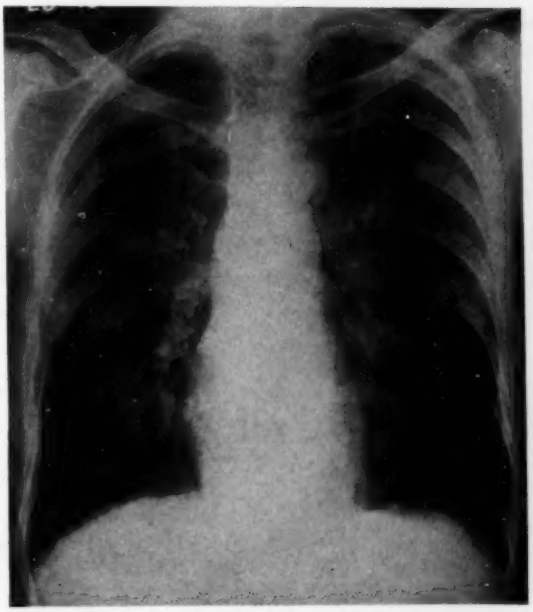

Fig. 7. Case IV.

Case IV. (P. C. U-390800) The patient was a man, aged sixty-three, who had been well until three weeks prior to admission. He entered the hospital complaining of severe headache. A deep chest cough productive of a half cup of foul sputum daily subsided after five days, leaving soreness in the lower dorsal spine and epigastrium. The pain was not related to eating but was aggravated by coughing. On admission the patient was cachectic and in severe pain, located chiefly in his back. Physical examination of the chest was negative. Biopsy of an enlarged supraclavicular node revealed metastatic adenocarcinoma. The patient retrogressed rapidly and died nine days after admisson.

Roentgenograms of the chest showed a slight, ill-defined enlargement of the hilar shadows and numerous fine miliary areas of increased density throughout both lung fields which were connected with each other by a fine, irregular network of linear markings, most pronounced in the central and lower portions of both lungs, but which also extended to the upper lungs and apices bilaterally (Fig. 7).

Anatomic Diagnosis. Scirrhous carcinoma of the stomach with metastases to the regional and supraclavicular lymph nodes, adrenals, lungs, liver and vertebral marrow.

At autopsy, a scirrhous carcinoma involved the entire circumference of the body of the stomach. The wall was thickened to 1 cm., the

serosal lymphatics were outlined by the tumor which they contained, and the lymph nodes along the lesser curvature were involved. Each pleural cavity contained approximately 350 cc. of serosanguineous fluid, and a few fine fibrous adhesions were found at each apex and base. The visceral pleura of both lungs was smooth and glistening, but the interlobular lymphatics

FIG. 8. Case v.

stood out as gray lines and along their course there were firm gray nodules, measuring as much as 1 cm. in diameter. On section, there was moderate congestion and edema, and the parenchyma was marred by prominent fibrous trabeculations. The trachea and bronchi were acutely inflamed and both bronchial and mediastinal lymph nodes contained tumor.

Microscopically, the primary tumor in the stomach was typically scirrhous in type with large numbers of anaplastic cells lying singly and in groups in a dense fibrous stroma. The tumor cells had completely destroyed the mucosa and infiltrated all layers of the organ. In the serosa they were found to lie predominantly in the perivascular lymphatic channels. All sections of the lung showed essentially the same microscopic picture. The metastatic tumor cells lay entirely within the lymphatics of the pleura, bronchial walls and around blood vessels. The intervening parenchyma was free from tumor and showed an early bronchopneumonia with mild congestion and edema. Many small pulmonary arteries were occluded by thrombi and some of these contained tumor cells at the level of section. Occasionally atypical cells were found in the capillaries of the alveolar walls.

CASE V. (C. M. U-397203) A man, aged seventy-three, entered the hospital complaining of constipation and abdominal pain and distention of three weeks' duration. The onset of his symptoms had coincided with the development of a "cold" characterized by a cough productive of foamy white sputum. For the past two days he had not defecated. A laparotomy with transverse colostomy was performed and metastatic tumor was found throughout the peritoneal cavity. Subsequently he developed tachycardia, rapid noisy respirations with cyanosis and dullness to percussion over the right lower chest. In spite of oxygen therapy his respiratory embarrassment continued until death on the tenth postoperative day.

Roentgen examination of the chest showed an irregular pattern of increased density with a linear design extending throughout both lungs from the hilum to the periphery, through which numerous small, irregular areas of increased density were scattered. These changes involved the lower portions of the lungs more than the apical regions. A small amount of fluid was present in each pleural sinus (Fig. 8).

Anatomic Diagnosis. Carcinoma of the body of the pancreas with metastases to the peritoneum, mesenteric and retroperitoneal lymph nodes, pleura, lungs, pericardium and liver.

At autopsy, the primary tumor was found in the body of the pancreas. It measured approximately 7 cm. in diameter and had produced atrophy of the parenchyma distal to it. The pleural cavities each contained roughly 300 cc. of fluid, and there were a few fibrous adhesions at the right apex. The outstanding gross finding in the lungs, apart from a moderate edema, was the presence of innumerable 2 to 4 mm. gray nodules, situated beneath the pleura and in immediate relation to the bronchi. In the left lower lobe there was an almost spherical tumor mass, 3 cm. in diameter. The trachea and bronchi showed slight mucosal congestion.

Microscopically, the pancreatic tumor was a fairly well differentiated adenocarcinoma, and sections taken from tissue in the immediate neighborhood showed cancer cells in the dilated perineurial lymphatics. The metastatic tumor in the lung was almost entirely limited to the pleural, bronchial, periarterial and perivenous

lymphatics, as well as the interlobular septa. The tumor cells retained their ability to form glands and in some fields were producing a colloid substance. In general, there was an acute bronchitis with minimal spread of the inflammation into the surrounding parenchyma. No blood vessel change could be detected apart from a few thrombosed arteries. No sections were taken of the mediastinal lymph nodes.

Case vi. (M. H. U-400685) The patient, female, aged fifty-nine, was under observation for eighteen months because of epidermoid carcinoma of the cervix. One month before death she coughed up a little blood and developed dyspnea and a dry hacking cough, both of which became increasingly severe. Fluid accumulated repeatedly in the left chest after tapping. On admission the patient was in extreme respiratory distress and slightly cyanotic with Cheyne-Stokes respiration. The pulse rate ranged from 100 to 150. A left pleural tap in the hospital was not followed by a reaccumulation of fluid, but the patient became more dyspneic, weaker, and died four days later.

Roentgenologic examination of the chest showed a rather diffuse, increased density involving the right lower lung field. A fine network of rather dense and broad trabeculation was visualized in the upper lung field where the lung appeared to be fairly well aerated. The left lung was clear (Fig. 9).

Anatomic Diagnosis. Epidermoid carcinoma of the cervix with adnexal extension, and with metastases to the lungs and pleura.

Postmortem examination of the cervix showed that it had been replaced by tumor which extended into the surrounding soft tissue. The left pleural cavity was partially obliterated by fibrinous adhesions in which there were many small granular tumor nodules. The right pleural cavity showed a fibrinous exudate over the posterior and medial surfaces. On section, the right lung was not remarkable apart from moderate edema. The left lung showed tumor nodules not only over its pleural surface but throughout the parenchyma. These nodules varied from 1 to 15 mm. in diameter. Their particular distribution was not noted. The bronchial lymph nodes on the left contained obvious metastatic tumor and several of the mediastinal nodes were also involved.

Microscopically, the cervical tumor was an undifferentiated epidermoid carcinoma (Grade 4). Its pulmonary metastases in the left lung were predominantly lymphatic so that small and large groups of atypical cells were found in the bronchial walls and around blood vessels. In a few fields the intervening parenchyma contained masses of tumor cells which had filtered into the alveolar spaces in a manner reminiscent of inflammatory cells in pneumonia; that is, without producing necrosis of alveolar walls. In addition, there was an early bronchopneumonia.

Fig. 9. Case vi.

Case vii. (L. T. U-417778) A man, aged forty-one, came to the hospital complaining of a productive cough, chest pain and increasing dyspnea of three months' duration. Physical examination showed fluid and consolidation in the right chest, a pulse averaging 110, clubbing of the fingers, cyanosis with a malar flush, generalized lymphadenopathy, enlargement of the liver and edema to the sacrum. Bronchoscopic biopsy revealed an undifferentiated carcinoma. Repeated right chest taps were performed as blood-stained pleural fluid rapidly accumulated. Each time less fluid could be withdrawn until finally only a few centimeters were obtained. Still his cyanosis and dyspnea increased. Latterly the sputum became blood streaked. The patient died nineteen days after admission.

Roentgenograms of the chest showed a moderate amount of fluid in the right pleural cavity. The right hilum appeared to be enlarged, and from it extended a string-like design of increased density, which involved the entire right lung but was more marked in its downward

than upward radiation. The trabeculated area extended to the periphery and was of uneven thickness and irregular pattern (Fig. 10).

Anatomic Diagnosis. Colloid carcinoma of the right lung with metastases to the pectoral region, mediastinal lymph nodes, pericardium, heart, pulmonary artery, aorta and adrenals.

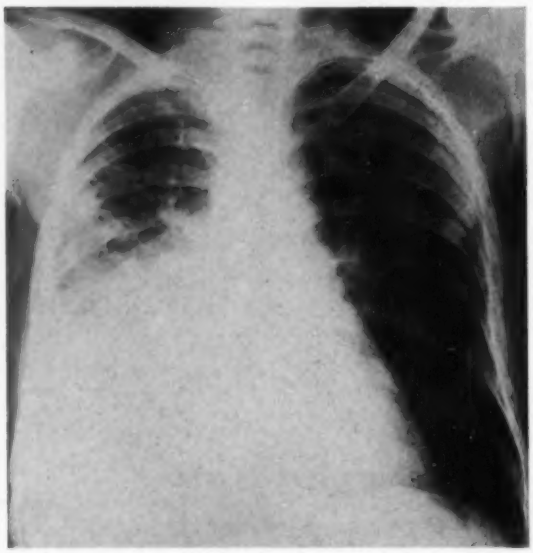

FIG. 10. Case VII.

At postmortem, it was difficult to determine the exact point of origin of the tumor in the lung. In the mucosa of the right main bronchus there were many pinhead-sized, firm nodules. The apex of this lung was covered with a greatly thickened pleural layer of cartilaginous consistency, while the rest of the pleura was roughened by a fibrinous exudate. Throughout the entire parenchyma of this lung the bronchi and blood vessels were surrounded by cuffs of gray, firm tissue, and many small nodules were found in the mucosa of the larger bronchi. The left lung showed collapse and edema in its lower lobe. The bronchial and mediastinal lymph nodes were almost completely replaced by tumor. Each pleural cavity contained approximately 500 cc. of blood-tinged fluid and the right was obliterated by fibrinous adhesions.

Microscopically, the tumor in the right lung was extremely anaplastic and for the most part the cells appeared in small groups without definite arrangement, although occasionally vague acini seemed to be forming. Many of the cells contained a colloid substance within their cytoplasm. Most of the sections showed extensive infiltration by tumor to which there was a pronounced fibroblastic response. However, in those areas which were relatively uninvolved, groups of tumor cells were found to be confined within the lymphatics of the bronchi and blood vessels. Furthermore, there were profound vascular changes similar to those described in Case II. In general, the parenchyma of the lung showed a moderate intra-alveolar hemorrhage with many pigmented phagocytes and a mild bronchopneumonia. The left lung was essentially normal. The spleen and kidneys were congested and the liver, which weighed 2,100 grams, showed central congestion and necrosis. These findings undoubtedly indicate some degree of right heart failure.

CASE VIII. (G. M. U-207642) A woman, aged fifty-nine, was treated for a year with radium and roentgen therapy because of a carcinoma of the cervix proved by biopsy. Three months prior to death she developed a chronic cough which gradually increased in severity. Shortly before death there was moderate respiratory

FIG. 11. Case VIII.

embarrassment. During the period of observation the roentgen appearance of the lungs changed from a questionable lesion to the characteristic appearance of lymphatic spread of carcinoma. The first roentgenogram of the chest showed a questionable increase in the linear markings throughout both lungs. Examination three weeks later showed some gross enlargement of both hilar shadows from which an ir-

regular, string-like design of increased density radiated downward and toward the periphery, and to a lesser extent upward. The regularity of the trabeculated network was broken up by numerous more nodular areas (Fig. 11).

The patient died at home and no autopsy was obtained.

Case IX. (C. B. HH 41-1060) A man, aged sixty-two, was treated for carcinoma of the tongue with very extensive metastatic involvement of the maxillary and cervical lymph nodes. While he was under observation, the characteristic appearance of lymphatic spread of cancer became evident on the roentgenograms of the chest. A diffuse uneven linear area of increased density, radiating from the hilar shadows toward the lung fields, most marked in the central and basal portions, was present at subsequent examinations. Between the linear markings a fine beaded network was visible due to numerous more nodular areas of increased density which appeared to fall within the primary pattern. A small amount of fluid was present in the right pleural cavity. At a final examination ten days later the process had increased in extent and intensity (Fig. 12).

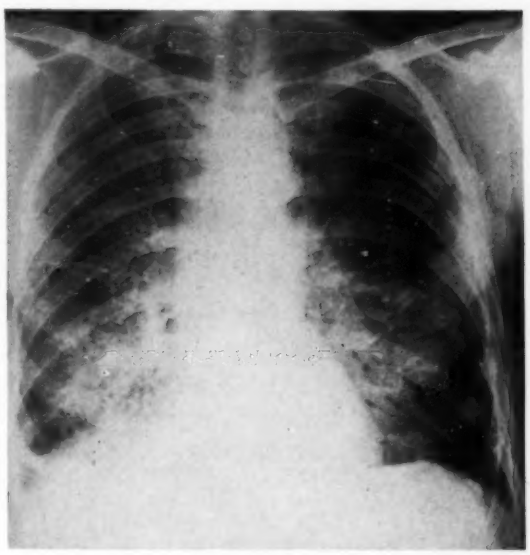

Fig. 12. Case IX.

The patient failed rapidly. A paroxysmal cough and respiratory embarrassment were marked for the two weeks before death, which occurred at home. No autopsy was obtained.

Case X. (H. N. U-392861) A man, aged thirty-one, was under treatment for melanotic sarcoma of the left arm with metastasis to the axilla. Four months before death the tumor was excised from the arm and the diagnosis confirmed microscopically. At this time there were no pulmonary symptoms and the roentgenogram of the chest showed a barely noticeable increase in the pulmonary markings with a possible suggestion of a nodular process involving both lungs homogeneously.

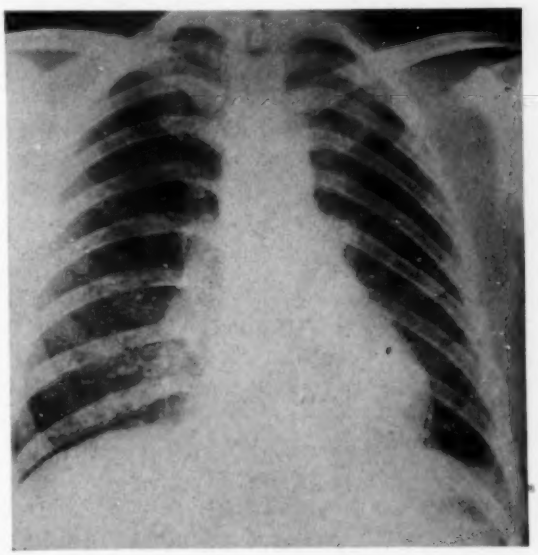

Fig. 13. Case X.

Three weeks later, an axillary dissection was carried out and the nodes were found to contain tumor. Roentgen examination showed a marked homogeneous miliary process in both lungs, due to irregular tiny nodules connected with each other by uneven linear areas of increased density (Fig. 13).

One month later the patient developed pain in the right chest, cough and fatigue. Roentgen examination disclosed further increase in the process. The patient died at home two months afterwards. No autopsy was obtained.

DISCUSSION

From these case reports it is evident that the roentgenologic appearance of lymphatic spread of carcinoma, although often difficult to interpret, is fairly characteristic. The best and most complete description is still that given by Assmann.[1] Roentgenograms show a diffuse, string-like formation

of increased density, which radiates from the hilum toward the periphery. The trabeculae break up into a fine network, scattered through which are numerous miliary nodules that usually correspond to the point of intersection of the trabeculae, or represent fine lymph vessels which are hit edge-on by the roentgen rays. The design in most cases is more marked in the central and basal portions of the lung, but it extends into the periphery and also into the upper portions. In only 1 of our cases (Case III, in the final stage) was the upper lung field more involved than the lower. Occasionally the changes are more marked on one side, or are unilateral. Often slightly larger nodules, due to large metastatic masses, are scattered between the linear markings and the fine miliary nodules. In most instances enlargement of the hilar nodes is obvious on the roentgenogram. Occasionally a small amount of fluid may be present in the pleural cavities.

In brief, the roentgen appearance is characterized primarily by a prominent linear trabecular network of increased density beneath which fine miliary nodules are visualized less distinctly. This is entirely different from the more common types of metastases which appear either as nodular or diffuse densities.

Lymphatic spread of cancer throughout the lungs represents the end-result of a carcinoma which usually originates in a distant organ but occasionally within the lung itself. In most instances, both lungs are evenly involved, but when the condition arises from an intrapulmonary tumor, or occasionally under other conditions, the lymphatic spread may occur on one side only or predominantly on one side. Anatomically there are two lymphatic systems within the lungs, one the deep intrapulmonary system and the other a more superficial pleural system, which drains the pleura and the most peripheral portion of the lungs. The intrapulmonary system extends through the bronchial and perivascular tissue and drains into the hilar lymph nodes. The pleural system runs in the connective tissue of the pleura and the interlobar septa, and also drains into the hilar lymph nodes, or to the thoracic duct. A sparse anastomotic circulation exists between these two systems at the periphery of the lungs.[11] It is predominantly involvement of the deeper intrapulmonary system which leads to the characteristic appearance of intrapulmonary lymphatic spread.

Two of our cases (Cases VI and VII) showed the process confined almost entirely to one side of the chest. In Case VII the primary tumor was a bronchiogenic carcinoma which led to a diffuse spread throughout the entire right lung. In Case VI, the chest examination disclosed changes in the right lung only, the lymphatics being invaded by metastatic tumor with unilateral involvement of the bronchial lymph nodes. The left side was practically free of disease. In another case (Case III) marked involvement of the right lung preceded the involvement of the left lung. The first roentgenogram showed the left lung to be practically free of disease, while re-examination one week later disclosed that the process had spread diffusely and evenly over both lungs.

The roentgen diagnosis of lymphatic spread of cancer is often difficult unless it is suggested by a known primary tumor or proved metastatic disease. In some cases, characteristic roentgenologic findings appear before the clinical symptoms; in a few instances, clinical signs and typical roentgen pulmonary changes are present with no suggestion of malignant disease elsewhere. Case 1 illustrates this latter observation. In this patient the diagnosis was not established before death despite the characteristic roentgenogram, which was interpreted as bronchiectasis and fibrosis because nothing pointed toward malignant disease. Only at autopsy was the small, silent carcinoma of the pancreas found, with widespread metastases and marked invasion of the pulmonary lymphatics.

With reference to the pathology of these cases, it might be well to say at the outset that nothing new or startling has been

found. Beginning with Andral[2] in 1829 a considerable number of reports have accumulated in the literature. These are predominantly of European origin and cover the subject rather thoroughly. It is not amiss, however, to emphasize certain points.

Case reports indicate that approximately 70 per cent of the cancers that give rise to pulmonary lymphatic spread have originated in the stomach. The remainder have arisen in the lung, breast, prostate, colon, gallbladder, tongue, kidney and ovary.[12,14,16] In our group of 10 cases, the primary tumor was situated as follows: 3 in the stomach, 2 in the pancreas, 2 in the cervix, 1 in a bronchus, 1 in the tongue, and 1, a melanotic sarcoma, first appearing in the left arm. With one exception the tumors were undifferentiated. Diffuse pulmonary lymphatic metastasis occurs about twice as often in men as in women, undoubtedly because of the high incidence of cancer of the stomach in the former. In this series, the same ratio obtained, 7 occurring in men and 3 in women.

In general, this diffuse infiltrative form of cancer occurs in younger persons than the average for cancer, the majority of cases occurring in the two decades thirty to forty-nine. In our group, the ages ranged from forty-one to seventy-three, with an average of fifty-nine years, which falls in the expected cancer age.

It is intriguing to speculate how and why these tumors spread to the lung and elsewhere in this particular fashion. It will be noticed from this report and others that an almost constant finding is involvement of the hilar lymph nodes, and furthermore, that there is a tendency to widespread lymph node involvement. If sections are taken from tissue adjacent to the tumor, lymphatic permeation is a more outstanding feature than in the average malignant tumor, and the perineurial lymphatics are frequently involved. Thus, there is a general preference for lymphatics. If one assumes that hilar lymph node involvement is a constant feature, cancer cells must reach these nodes by way of their afferent or efferent lymphatics. If dissemination is via the afferent route, the lungs must be involved first. This supposition would necessitate primary blood spread or pleural metastasis with final invasion of the pulmonary lymphatics; this seems highly unlikely and unnecessarily complicated. The alternative route is favored by most authors;[9,12,16,17,18] that is, retrograde permeation of the pulmonary lymphatics following involvement of the higher lymph nodes. This theory seems reasonable because the lymphatic circulation ebbs and flows sluggishly with little pressure. When one path of drainage is blocked, the flow is reversed and the fluid is carried away along neighboring channels. A similar phenomenon occurs in sarcoidosis in which hilar lymph node enlargement seems to be the primary manifestation. If the lung parenchyma becomes involved, the lesions develop as a fan-shaped radiation from the hilum.[5,6]

The pathologic appearance in the lungs of the autopsied cases exhibited the constant picture of lymphatic permeation with variation in the form of minor or major vascular change. On gross examination the lungs were voluminous, while the pleura was smooth and seldom marred by a fibrinous exudate. The pulmonary lobules were outlined by the gray lines of dilated lymphatics which had formed a network suggesting a mosaic pattern. Nodules often formed at the angles of this network. The cut surfaces were remarkable in that each bronchus or blood vessel seemed to be surrounded by a gray cuff of tumor, and here again the lymphatics were prominent. In general the parenchyma was edematous. The hilar lymph nodes contained gross tumor in all but one case (no microscopic sections were taken in this case), and in all instances there was a variable quantity of pleural fluid.

Microscopic sections showed that the lymphatics throughout the entire lung were dilated and contained groups of metastatic tumor cells, some of which were necrotic. They were found in the lymphatics of the

pleura, the thickened interlobular septa, around blood vessels of all sizes and throughout the bronchial walls. Invasion of the bronchial mucosa had occurred at various points in all cases. In each case, also, a very few tumor cells lay scattered in the alveoli, and in one instance they had filled these spaces over a few low power fields in the manner of inflammatory cells in pneumonia without as yet producing tissue destruction.

Widespread and profound arterial damage had taken place in Cases II, III and VII. The changes have not been explained satisfactorily. The tumor cells in the lymphatics of the adventitia seemed to have excited an intense fibroblastic reaction so that the coat was greatly thickened. The media and intima often contained small nests of tumor cells. This invasion by tumor cells was frequently accompanied by a lymphocytic infiltration. In many fields the intima of the small arteries and arterioles was thickened by a layer of acellular fibrous tissue in which the fibers were widely separated, presumably by fluid. This was either concentric or plaque-like, and occurred in the presence or absence of tumor cells in the coat itself. Furthermore, tumor cell emboli, usually associated with thrombotic material, were present throughout the arterial system. In the small radicles this had produced occlusion, and in some instances partial or complete organization with recanalization had occurred. In the large arteries, there was no occlusion, but a few vessels were found where the tumor cells and thrombotic material were adherent to the intima from which organization and endothelialization of the mass were taking place. Tumor cells had also reached the alveolar capillaries. In Case IV, tumor emboli were present in the vessels without other arterial change, and in Case V, arterial thrombosis was found without tumor emboli or arterial change. The pulmonary veins, on the other hand, contained neither thrombi nor tumor emboli, although the surrounding lymphatics were dilated and partially filled with tumor cells. Occasionally, however, fibrous intimal thickening and tumor cell invasion of the outer layers of the venous wall were present. Both arteries and veins were often compressed by the surrounding dilated lymphatics.

The reason for the arterial changes, which were first described by Girode[7] in 1889, is not entirely clear. Wu,[18] in a beautifully illustrated paper, states his belief that the intravascular deposition of carcinoma plays an important part in the genesis of the obliterative changes and that the edematous thickening of the arterioles is a local manifestation of the general interstitial edema of the lung following obstruction. Greenspan[8] is of the opinion that the same fibroplastic factor that is responsible for the extensive increase in the perivascular connective tissue might also be responsible for the intimal hyperplasia. From the present observations, we are inclined to believe that (1) tumor cell embolism by way of the blood stream is the sole factor leading to the arterial occlusion (if other factors are present, thrombosis should also occur in the veins); (2) the adventitial thickening is initiated by the presence of tumor cells; (3) if the intimal thickening is a local manifestation of edema, it should occur equally in the arteries and veins and should appear more often than it does in lymphangitic carcinosis. In our series, intimal thickening occurred only in those cases which showed a combination of tumor cell invasion of the blood vessel walls and thrombosis of the arterial lumina. We believe that the two must be related.

The major symptoms in lymphatic carcinosis of the lung occur with great regularity and contrast sharply in their severity with the negligible physical signs. In the various cases reported throughout the years the paucity of physical findings in the lungs has been emphasized again and again, and the present report does not refute the reiteration. The pulmonary symptoms, however, often overshadow those which arise from the primary tumor, usually throughout the entire, brief course of the patient's illness. The important clinical findings are dysp-

nea, cyanosis, a productive cough and rapid cachexia. Dyspnea occurred in 6 of the 7 autopsied cases, and in these patients it was an outstanding feature of the terminal picture and did not respond to the usual palliative measures. Cyanosis was present in 4 patients, and all the patients had varying amounts of sputum. When the clinical and pathologic findings were suitably tabulated in each case it seemed that no single anatomic abnormality could account for any given symptom. The variety and severity of the pathologic findings seem to explain adequately the clinical manifestations. All the patients had a productive cough and in 3 of them the sputum was blood streaked. This is accounted for by the destruction of the bronchial mucosa by invading tumor cells, a phenomenon encountered in all the lungs examined. King and Castleman[10] studied 109 cases of pulmonary metastatic tumor and found that 20 (18.5 per cent) showed bronchial invasion either by direct extension from a neighboring tumor or by lymphatic spread. Four of the 20 patients raised blood-streaked sputum; 9 had no pulmonary symptoms.

It is interesting that a certain number of patients with lymphangitic carcinosis die with definite clinical signs of right heart failure, and at autopsy the right ventricle shows hypertrophy and dilatation. Two of the more recent reports concerning this mode of death are those of Greenspan[8] and Brill and Robertson.[4] Arterial changes were present in roughly half of the cases reported in the literature. In the clinical records of our patients, heart failure was not mentioned, though all but Case IV had tachycardia (between 120 and 130), and in Case VII there was liver enlargement and peripheral edema. No electrocardiographic tracings were made. None of the patients examined was said to have had right ventricular hypertrophy, but unfortunately this abnormality is overlooked rather easily. One case* with cardiac change has been encountered in this laboratory, but it was not included in the present series because pleural fluid obscured the roentgen pattern. This patient had cancer of the stomach with lymphatic spread to the lungs without endarteritic changes in the pulmonary vessels. There were clinical signs of right-sided heart failure and right axis deviation in the electrocardiographic recording.

Only one patient (Case IV) showed significant blood changes, and these were anemia and purpura. Furthermore, this patient was the only one in whom bone marrow metastases were found. Jarcho,[9] in 1936, reviewed the subject of diffuse infiltrative carcinoma. From his study he concluded, not without good reason, that lymphangitic carcinoma of the lung and metastatic carcinoma of the bone marrow associated with thrombopenia and purpura tend to occur in the same type of case and in the same individual. His patients were predominantly young adults with a scirrhous or diffusely infiltrating gastric carcinoma which was often silent clinically, and whose illness pursued a rapid, retrogressive course. If the bone marrow was heavily involved, the clinical appearance was that of a blood dyscrasia. If the lungs were extensively involved, respiratory symptoms attracted the greatest attention. The question arises whether the tumor cells reach the bone marrow by way of the blood stream or by the lymphatic channels. Maximow and Bloom[13] say that lymphatics have not been demonstrated in the bone marrow, but the interesting experiments of Kolodny[11] cast doubt on this statement.

In correlating the roentgenologic manifestations with the pathologic findings it seemed evident that the presence of masses of tumor cells within dilated lymphatics around bronchi and blood vessels was responsible for the peculiar roentgenologic pattern. Since the roentgen findings were similar in all cases it is believed that the microscopic severe arterial damage and tumor cell invasion of the bronchial lumina played a negligible part in the formation of roentgenologic appearance. Infiltration of alveolar spaces by tumor cells may account

* Case Records, Massachusetts General Hospital, Case 29291. *New England J. Med.*, 1943, *229*, 180.

for the larger densities which were occasionally found on the roentgenograms and which were difficult to explain solely on lymphatic involvement. Fibrosis, pulmonary edema, and blood vessel congestion, which under certain conditions may resemble the roentgenologic appearance of lymphatic spread, were not found in any of our autopsied cases to the extent that could produce visible changes on the roentgenogram.

DIFFERENTIAL DIAGNOSIS

Despite the fairly definite roentgen appearance of lymphatic spread, a number of other conditions resemble it very closely. Foremost among the possible differential diagnoses is *miliary tuberculosis*. Both clinically and roentgenologically the two conditions resemble each other. Cyanosis, severe dyspnea and fever may occur in both. The age group in miliary tuberculosis is usually, but not always, younger. Roentgenologically, there are certain differential points. In miliary tuberculosis, the hilar shadows are as a rule less dense, and enlargement, if present, is less marked; the string-like design so characteristic of lymphatic metastasis is not present, but instead the fine nodular shadows scattered over the lung represent the typical appearance. The upper portions of the lungs are usually involved in miliary tuberculosis while they are less apt to be involved in metastatic malignancy.

Pulmonary congestion and edema may also present a similar diffuse string-like design roentgenologically, due to congestion in the blood vessels. The lung fields are often hazy because of edema; the blood vessels tend to become smaller toward the periphery. In lymphatic spread, on the other hand, the characteristic design is more uneven, the lymphatics growing smaller in certain areas and in others becoming thicker toward the periphery. Roentgenoscopic examination of the heart may be helpful in some cases as a differential procedure. Clinically dyspnea, respiratory embarrassment and cyanosis are common in both conditions.

Pneumoconiosis, in contrast to lymphatic spread of cancer, presents, as a rule, a much denser roentgen appearance with more circumscribed, harder nodules. Some forms of pneumoconiosis, especially the acute type, may be extremely difficult to differentiate, but these cases are fairly rare, and the history of exposure may help in the diagnosis when other differential points fail.

In some cases of *sarcoid* the involvement of the lungs simulates the roentgenologic appearance of lymphatic spread of cancer. However, in sarcoid the involvement of the hilar lymph nodes is usually characterized by sharply defined masses which are present equally on both sides. Enlargement of the spleen may give confirmatory evidence in some cases, and as a rule the marked roentgenologic involvement is in great contrast to the minimal clinical manifestations.

Other conditions which must be ruled out are primary *fibrosis with emphysema*, and *bronchiectasis* as in Case 1. In these two conditions a long history dating back farther than would be the case in lymphatic metastatic malignancy is usually elicited.

In addition to the preceding differential possibilities, there have come under our observation recently some cases of *atypical pneumonia*, in which the roentgen appearance in its resolving stages somewhat resembles lymphatic spread of cancer. The clinical history of a preceding upper respiratory infection would, however, establish the diagnosis of pneumonia.

Although, as a whole, the roentgenologic findings are quite typical and can be distinguished from other conditions by careful study, in the individual case a definite diagnosis may be difficult unless other roentgenologic or clinical evidence of malignant disease exists.

SUMMARY

Ten cases of lymphangitic carcinosis of the lungs, seen in recent years at the Massachusetts General Hospital, are presented to illustrate the lymphatic spread of cancer. Autopsy was performed on seven of the group.

The roentgenologic appearance presents a typical pattern which is entirely different from that of the usual pulmonary metastasis. It is characterized by a network of increased density, radiating from enlarged hilar lymph nodes and trabeculating through the lung fields in an uneven manner, and is interspersed by numerous fine nodules. It is more marked in the lower and central portions of the lungs and occasionally may be unilateral. Differential diagnoses of miliary tuberculosis, pulmonary edema and congestion, sarcoid, pneumoconiosis, and bronchiectasis, among others, must be ruled out.

The microscopic picture in the lungs is characterized by widespread permeation of the lymphatics by tumor cells and, in a certain number of instances, profound arterial damage. Anaplasia of the tumor, rather than its primary site, appears to be the important factor in this form of dissemination.

The age and sex incidence, the mode of spread and the arterial changes are discussed.

The common clinical findings are dyspnea, cyanosis, productive cough and rapid cachexia. Pulmonary signs are minimal; but pulmonary symptoms overshadow those which arise from the primary tumor.

REFERENCES

1. Assmann, H. Die klinische Röntgendiagnostik der inneren Erkrankungen. Fourth edition. F. C. W. Vogel, Leipzig, 1929.
2. Andral, G. *Précis de path. anat., Paris*, 1829, ii, 444.
3. Bard, L. Du diagnostic de la lymphangite pulmonaire cancéreuse généralisée. *Rev. méd. de la Suisse Rom.*, January, 1918. Quoted by Schwarzmann.[14]
4. Brill, I. C., and Robertson, T. D. Subacute cor pulmonale. *Arch. Int. Med.*, 1937, *60*, 1043–1057.
5. Bruce, T., and Wassen E. Clinical observations on course and prognosis of lymphogranulomatosis benigna Schaumann, particularly in regard to pulmonary lesions. *Acta med. Scandinav.*, 1940, *104*, 63–104.
6. Case 30331. Case Records Massachusetts General Hospital, *New England J. Med.*, 1944, *231*, 268.
7. Girode. *Arch. gén. méd.*, 1889, *1*, 50.
8. Greenspan, E. B. Carcinomatous endarteritis of pulmonary vessels resulting in failure of right ventricle. *Arch. Int. Med.*, 1934, *54*, 625–644.
9. Jarcho, S. Diffusely infiltrative carcinoma; a hitherto undescribed correlation of several varieties of tumor metastasis. *Arch. Path.*, 1936, *22*, 674–696.
10. King, D. S., and Castleman, B. Bronchial involvement in metastatic pulmonary malignancy. *J. Thoracic Surg.*, 1943, *12*, 305–315.
11. Kolodny, A. Relation of bone marrow to lymphatic system; its rôle in spreading of carcinomatous metastases throughout the skeleton. *Arch. Surg.*, 1925, *11*, 690–707.
12. Lumsden, C. E. Pulmonary carcinoma; pathological study of series of cases with special reference to route of spread and to factors that determine mode of spread. *Glasgow M. J.*, 1939, *131*, 57–72.
13. Maximow, A. A., and Bloom, W. Textbook of Histology. W. B. Saunders Co., Philadelphia, 1942.
14. Schattenberg, H. J., and Ryan, J. F. Lymphangitic carcinomatosis of lungs; case report with autopsy findings. *Ann. Int. Med.*, 1941, *14*, 1710–1721.
15. Schmidt, M. C. Die Verbreitungswege der Karzinome und die Beziehung generalisierte Sarkome zu den leukämischer Neubildungen. G. Fischer, Jena, 1903.
16. Schwarzmann, A. Ueber generalisierte carcinomatöse Lymphangitis der Lungen. *Acta radiol.*, 1934, *15*, 491–501.
17. von Meyenburg, H. Zur Kenntnis der Lymphangitis carcinomatosa der Lungen und Pleuren. *Cor.-Bl. f. schweiz. Aerzte*, 1919, p. 1668. Quoted by Schwarzmann.[14]
18. Wu, T. T. Generalised lymphatic carcinosis ("lymphangitis carcinomatosa") of lungs. *J. Path. & Bact.*, 1936, *43*, 61–76.

PULMONARY CHANGES IN CARDIOSPASM

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TWO cases of severe cardiospasm are presented which demonstrate extensive pulmonary changes in the roentgenograms, yet presented no significant pulmonary signs or symptoms. One case, after six years of observation, had a short episode of fever, cough, dyspnea and hemoptysis which was diagnosed as influenza. At no other time was there evidence of cough, dyspnea or auscultatory abnormalities with either patient. Their complaints were limited to manifestations of esophageal obstruction and distention.

In previously reported cases of cardiospasm with changes in the chest roentgenograms, there have been associated cough, pain, dyspnea, etc. In such cases, the patients had ailments referable to pulmonary disease as well as to cardiospasm.

FIG. 1. Case 1. 1935. Moderate fibrosis of both lung fields, most prominent in the right upper lobe.

CASE REPORTS

CASE 1. In 1935, a male, aged fifty-seven, was admitted as an out-patient to the Mary Fletcher Hospital, Burlington, Vermont, for roentgen examination of the esophagus and stomach. He gave a history of intermittent difficulty in swallowing and occasional regurgitation of food.

Roentgen examination showed the esophagus slightly dilated, terminating below in a smooth, rounded defect, and a diagnosis of achalasia (cardiospasm) was made.

Two years later, he was admitted to the same hospital with similar complaints. He stated that his throat would feel filled after a few swallows of food or liquid and he would have to stop eating. He would lose considerable weight during these periods, then gain for a short time. During the month prior to admission, he had lost 14 pounds. There had been no cough and only one attack of vomiting.

Physical examination of the chest disclosed no abnormalities. Roentgenoscopy and roentgenography demonstrated slightly increased dilatation of the esophagus over that previously noted. Both lung fields (Fig. 1) showed evidence of a moderate amount of fibrosis, most prominent in the right upper lobe and in the vicinity of the interlobar septa on both sides.

The cardia was dilated, with partial relief of symptoms.

In 1941, he re-entered the hospital, weighing only 95 pounds. Again (Fig. 2), chest roentgenograms showed a marked increase in linear markings in all portions of both lungs with a distribution suggesting diffuse interstitial fibrosis. Bougies were passed, and the patient gained 11 pounds during the following month.

His final admission was in March, 1943. Three weeks prior to admission, he had had

"flu" with a temperature of 101° F. and slight hemoptysis. For one week, he had regurgitated several times a day. On admission, he was free from cough, expectoration and fever, and physical examination of his chest revealed no abnormalities. A roentgenogram of the chest showed no change. Esophagoscopy and dilatation were done, and the patient was subsequently discharged improved.

Comment. A man, aged fifty-seven, was followed for six years after cardiospasm was proved by roentgen examination. During this time, the lungs showed evidence of increasing fibrosis, apparently interstitial. He had one brief episode of cough and hemoptysis. Physical examination of the chest never showed any abnormality.

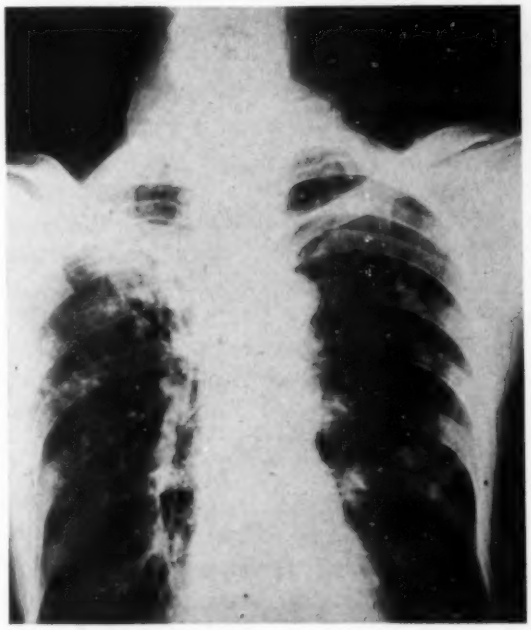

Fig. 2. Case 1. Six years later.

Case 11. A soldier, aged thirty-one, a former mechanic, had noted, over a period of four years, gradual and progressive difficulty in swallowing. Food seemed to stick in the upper part of the esophagus and at times he felt as if it would shut off his breathing entirely. Liquids aided the emptying of the esophagus. He would hiccup frequently after eating. Occasionally he would awaken at night to find his mouth filled with regurgitated food and fluid. He never choked on this material and, after spitting it out, could go back to sleep.

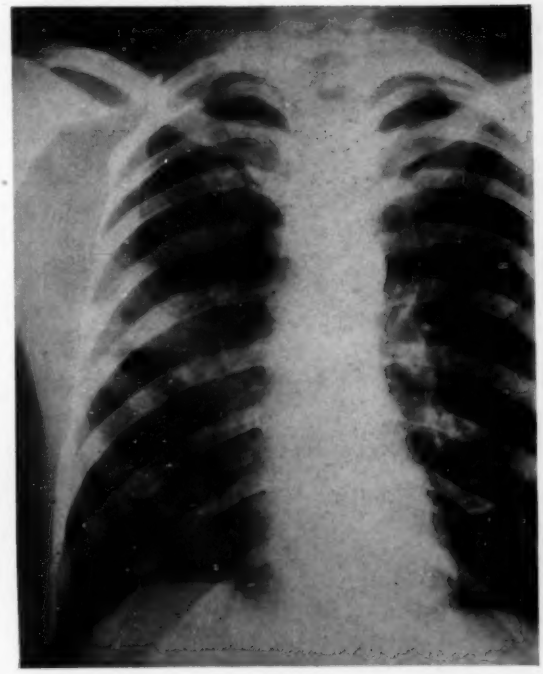

Fig. 3. Case 11. 1940. Note the large area of hazy density against the horizontal fissure on the right.

Three years before, the diagnosis of cardiospasm was established roentgenologically in a civilian hospital. Figure 3 is a posteroanterior roentgenogram of the chest taken at that time. Above and resting upon the horizontal fissure between the right upper and middle lobes there is a hazy area of increased density.

The patient had no pulmonary symptoms. A dilatation was performed with temporary relief.

At present, roentgenoscopy shows a dilated esophagus which is slightly elongated and which shows conical narrowing toward the cardia. Roentgenogram of the chest shows the same area of increased density in the right upper lobe (Fig. 4). On rotating the thorax under the roentgenoscope, this area is shown to lie in the parenchyma, but touching the lateral parietal pleura. The pleura is not demonstrably thickened over it and no cavities are seen.

Bronchoscopy and lipiodol filling of the bronchial subdivisions of the right lung showed no abnormality of the mucosa or size of the bronchioles.

Comment. A soldier with a history of four years of cardiospasm showed an area of increased density in the right upper lobe which persisted over a period of three years. It was thought to represent an area of fibrosis which developed from inhalation pneumonitis.

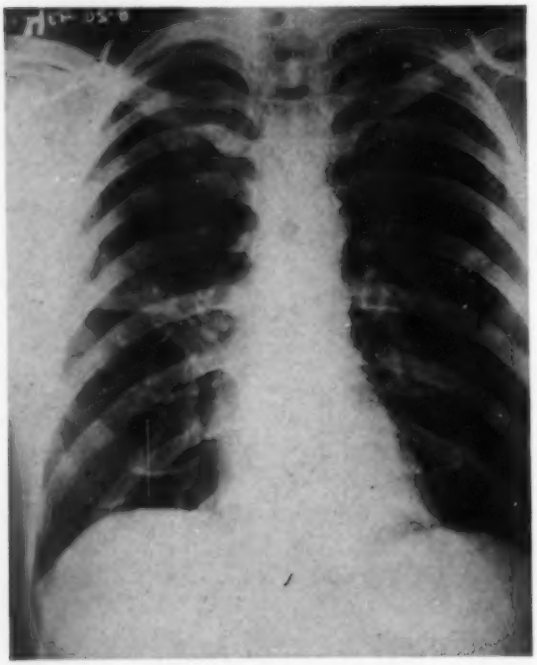

Fig. 4. Case II. Three years later. The area has not changed in size or shape.

ETIOLOGY OF PULMONARY CHANGES

Patients with cardiospasm are bothered frequently by coughing[4] and occasionally by sudden paroxysms of strangulation.[1,14] This is more often precipitated or aggravated by lying down, or may occur during sleep.[5] At that time, with the esophagus nearly horizontal, the large reservoir of food and fluid may spill into the pharynx and be aspirated. If asleep, the patient may be aroused by severe coughing. He may find his mouth and throat filled with fluid and food which will take several seconds to fully clear away. At times, however, this fluid descends farther into the bronchi and may lead to pulmonary disease.

Four types of pulmonary changes (roentgenologically) have been reported: basal pneumonitis, lung abscess, bronchiectasis and pleural effusion.

Basal Pneumonitis. Basal pneumonitis as a complication of cardiospasm was reported first in 1914 by Eisenstein[3] as double pneumonia. Mallory[6] reported the case of a man, aged sixty-four, who had suffered with cardiospasm for fifteen years; cancer developed at the lower end of the esophagus, leading to obstruction and death. "At the right lung base there was pneumonia or consolidation with many abscess cavities within the area." This was called an inhalation pneumonia.

The second case reported by Mallory[7] was a sixty-two year old woman who had had cardiospasm for forty-one years. A roentgenogram taken twenty-three days before death showed dullness at both bases posteriorly which was thought to be an aspiration pneumonia. However, at the time of autopsy, the lungs were clear.

The fourth case was seen by Schmidt.[11] A six year old girl was admitted with a history of repeated pulmonary infections over a period of two years, associated with fever. There were moist râles at both bases. With relief of cardiospasm by bouginage, the pulmonary symptoms ceased.

Lipoid pneumonia at both bases as a complication of cardiospasm was described by Thomas and Jewett.[12] A man, aged thirty-two, had suffered from cardiospasm for four years. In the recent months, cough and fever had been recurrent and râles were present at both bases. A roentgenogram of the chest revealed a hazy, finely mottled consolidation throughout the right lung and at the left base. Microscopic section of the lungs removed at autopsy showed numerous globules of fat scattered throughout patchy areas of consolidation in the bases. The fat probably originated from a high milk and cream diet that had been taken during the past months.

Lung Abscess. This has been reported twice in the literature as a complication of

cardiospasm. In 1927, Vinson[13] had a forty-two year old male patient who, after ten years of cardiospasm, developed cough with foul bloody sputum and fever. A large abscess cavity was found in the right upper lobe by roentgen examination. After esophageal bouginage, the pulmonary abscess cleared rapidly. A similar case was seen by Sampson.[9] Symptoms had been present for several months. On admission, the roentgenogram showed consolidation in the right middle lobe. He was treated conservatively and symptoms disappeared. In the course of the next three months, however, a cavity developed in the right middle lobe, which remained without causing symptoms for six years. A pneumolith rolled about in the bottom of this cavity. The patient, meanwhile, had no symptoms and the cardiospasm had been completely relieved. The long duration of the cavity without surrounding pneumonitis is noteworthy.

Bronchiectasis. The third pulmonary complication of cardiospasm is bronchiectasis. In 1933, Reeke[8] reported the case of a female, aged nineteen, who had been coughing for several years and showed clubbing of the fingers and toes. Study was concentrated on the lungs and bronchiectasis was demonstrated roentgenographically on both sides. Later cardiospasm was found which was so severe that gastrostomy was performed. Pneumonia ensued and the patient died. Autopsy confirmed the diagnosis of cardiospasm and bilateral bronchiectasis.

In the second case, reported by Schrire,[10] cough had been present for six years, with frequent attacks of pneumonia. At the age of twelve, bronchiectasis was discovered in the right middle lobe. One year later, a second bronchogram showed spread of the bronchiectasis into the right lower and left upper lobes. Six months later, cardiospasm was found for the first time and a bougie passed. There was immediate gain in weight and complete cessation of cough. One year after bouginage, bronchoscopy showed no disease. Relief of the cardiospasm had stopped the repeated trickling of the esophageal fluid into the trachea and bronchi. This was followed by healing of the chronic pulmonary disease. In other words, this may be a case of so-called "reversible bronchiectasis." Removal of the focus allowed the disease to regress.

Pleural Effusion. Chandler[2] reported the case of a female, aged fifty-one, who had had difficulty in swallowing for several months. She was seen first with a large pleural effusion and gave a history of transient chest pain five weeks before. One month later, cardiospasm was diagnosed and atropine given. The difficulty in swallowing and effusion disappeared simultaneously.

DISCUSSION

To these four complications of cardiospasm, we have added 2 cases of probable interstitial pulmonary fibrosis. In both cases, the lesions were observed over several years without change. Bronchoscopic and bronchographic studies of one patient revealed no abnormalities. The patients were asymptomatic from the pulmonary changes, although they suffered greatly from cardiospasm. The etiology of pathological condition in the lung is apparently inhalation of esophageal contents with pneumonitis and consequent fibrosis.

SUMMARY

Two cases of cardiospasm are reported with long-standing asymptomatic pulmonary lesions, probably interstitial fibrosis following aspiration pneumonitis. Attention is called to certain other pulmonary and pleural complications, some of which may be relieved by treatment of the primary condition.

REFERENCES

1. Ball, R. P., and Crump, A. C. Mega-esophagus (cardiospasm). *Radiology*, 1941, *36*, 575–582.
2. Chandler, F. G. Achalasia, with pleural effusion, simulating mediastinal growth. *Lancet*, 1938, *2*, 398.

3. Eisenstein, A. Beiträge zur Radiologie der Speiseröhre. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1914, *21*, 381–405.
4. Freeman, E. B. Symptoms, diagnosis and treatment of chronic cardiospasm. *Rev. Gastroenterol.*, 1940, *7*, 385–390.
5. Geppert, F. Cardiospasmmus und die spindelformige Erweiterung das Speiseröhre. *Centralbl. f. d. Grenzgeb. d. Med. u. Chir.*, 1914, *18*, 149–187.
6. Mallory, T. B. Cardiospasm; esophageal dilatation, marked. (Cabot case 19452.) *New England J. Med.*, 1933, *209*, 962–964.
7. Mallory, T. B. Dilatation and muscular atrophy of esophagus; cardiospasm. (Cabot case 24141.) *New England J. Med.*, 1938, *218*, 607–610.
8. Reeke, T. Angeborene doppelseitige Bronchiektasen mit Megaoesophagus. *Deutsche Ztschr. f. Chir.*, 1933, *241*, 488–494.
9. Sampson, D. A. Lung abscess due to esophageal overflow. *New England J. Med.*, 1938, *219*, 982–985.
10. Schrire, T. Bilateral bronchiectasis with cardiospasm. *Lancet*, 1938, *2*, 1225–1226.
11. Schmidt, H. W. Cardiospasm in a 6 year old child. *Proc. Staff Meet., Mayo Clin.*, 1936, *11*, 767–768.
12. Thomas, W. S., and Jewett, C. H. Pneumonia following the aspiration of fats from esophagus dilated as the result of cardiospasm. *Clifton Med. Bull.*, 1926, *12*, 130–138.
13. Vinson, P. P. Cardiospasm complicated by pulmonary abscess. *Am. J. Surg.*, 1927, *2*, 359–361.
14. Vinson, P. P. Cardiospasm. *Am. J. Surg.*, 1942, *56*, 79–86.

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CONGENITAL APLASIA OF THE LUNG

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THE antemortem diagnosis of agenesis of the lung with bronchographic follow-up for an interval of seven years is sufficiently rare to warrant addition to the roentgenological literature.

Madigan[8] reported 36 proved cases with postmortem findings of each. Van Loon and Diamond[10] recorded 40 genuine cases. Hurwitz and Stephens[6] describe an additional case and review 34 authentic cases adequately recorded in the literature. Additional solitary cases are recorded by Yampolsky and Fowler,[11] Elward,[4] Stokes and Brown,[9] and recently De Weese and Howard,[3] as well as many others in the foreign literature. The sum total did not appear to exceed one hundred proved cases, with a great majority of these representing postmortem findings and observations.

CASE REPORT

E. B. The patient was first seen at the chest clinic of the Cincinnati General Hospital on March 27, 1935, because of a tuberculous contact with an aunt. He had always been underweight and somewhat underdeveloped, now weighing 31 pounds (average weight 38 pounds). There is a past history of frequent colds, measles, pertussis, and an episode of pneumonia when he was eight years of age. Except for the tuberculous aunt the family history is negative.

Physical examination at this time shows marked contraction of the left hemithorax with a flat percussion note and diminished breath sounds except at the left apex. No râles were heard. The right chest was clear. A roentgenogram showed the entire left hemithorax to be opaque with the heart located near the left lateral chest wall. Two follow-up roentgenograms over a period of five years showed no significant changes.

The patient was next seen in the Children's Hospital where bronchograms were done (Fig. 1) after an episode of bronchopneumonia at the right base had subsided. He apparently was in good health during this interval until December, 1941. At this time he was again seen in the chest clinic at the Cincinnati General Hospital where no subjective complaints were elicited except that he failed to gain weight (55 lb.—average 67 lb.). P. P. D. No. 1 and No. 2 have been consistently negative over this period of observation. Bronchograms under bronchoscopic control were again done and the same findings in the left main stem bronchus, mediastinal shift to the left, and compensatory emphysema on the right were again demonstrated (Fig. 2).

The bronchoscopist (Dr. Sam Iglauer) reported that the left main stem bronchus was found to be 1.5 cm. in length, ending abruptly with a perpendicular carina and two small

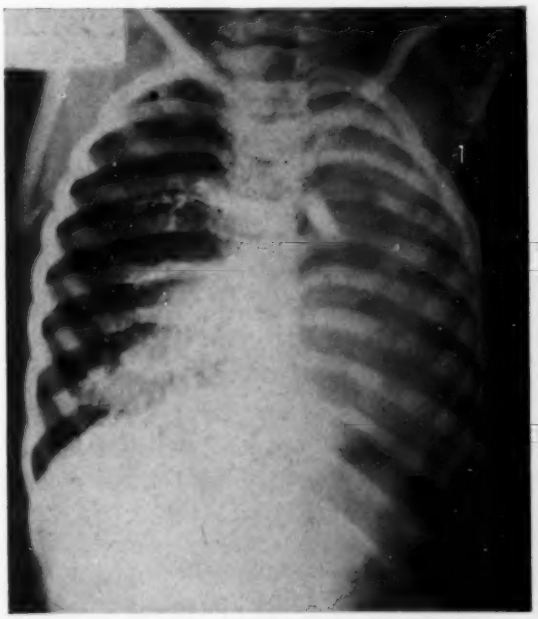

FIG. 1. March, 1935. Bronchogram showing the blunt filling of the left main stem bronchus with lipiodol. Homogeneous opacity of the entire left lung, with the trachea, heart and mediastinum shifted to the left. The left diaphragm is outlined by the distended fundus of the stomach. The right lung shows a moderate degree of emphysema. (Courtesy of Dr. William M. Doughty, Children's Hospital, Cincinnati, Ohio.)

FIG. 2. January, 1942. Follow-up bronchogram showing no significant change after an interval of seven years.

bronchi, 2 mm. in diameter. In his opinion, this represented a congenital failure in the development of the left bronchial tree and lung.

The latest roentgenogram, taken when the patient had another episode of pneumonia at the right base in March, 1943, still demonstrated no change in the findings relative to the left lung.

DISCUSSION

Clinically this congenital anomaly is asymptomatic. The general development is good or slightly delayed for the age of the patient. There may be slight flattening and diminished movement of the affected side. The heart and mediastinal structures are displaced to the side of the absent lung with the apical impulse being pronounced. Flatness and dullness are usually present to percussion while the breath sounds are absent or suppressed on auscultation. The left lung has a greater incidence of aplasia than the right in the ratio of two to one. The presence of an aplastic lung is consistent with a normal life if the patient survives the first year of life. One patient lived to the age of seventy-two, dying of a cerebral vascular accident.[4]

The principal roentgen findings show the displacement of the heart and mediastinal structures to either side with slight elevation of the ipsolateral diaphragm and narrowing of the intercostal spaces on the affected side. The contralateral lung usually shows increased aeration, indicating a significant degree of compensatory function.

Paralysis of the diaphragm from whatever mechanism, congenital absence of the diaphragm, diaphragmatic hernia and intrabronchial foreign body are to be considered in the differential diagnosis. Bronchoscopy and bronchography are the two important procedures in establishing the clinical diagnosis by excluding the above-mentioned conditions.

Arey,[1] in his discussion of the embryonic development of the respiratory system, says: "A groove-like evagination arises on the ventral side of the esophagus in the 3 mm. embryo. From the enlarged posterior ends of the groove two small lung buds grow out. Later in the development, the trachea and esophagus become separated by a constriction interrupted at the cephalic end of the larynx which is indistinguishable at the end of the fifth week. Muscle fibres and cartilaginous rings differentiate from the surrounding mesenchyme at the end of the seventh week. In a later metamorphosis of the lung buds, hollow evaginations grow out into the envelop of the connective tissue, enlarge and continue to branch, producing a tubular system. From the fine terminal tubules arise small outgrowths which constitute the pulmonary alveoli." The etiology of the aplasia is obscure. The most rational explanation for aplasia of the lung ascribes the condition to a developmental error of endogenous origin, perhaps primary in the pulmonary vascular system.

SUMMARY

A case of congenital aplasia of the left lung with bronchographic follow-up over a

period of seven years is presented together with a review of the literature.

REFERENCES

1. Arey, L. B. Developmental Anatomy. Third edition. W. B. Saunders Co., Philadelphia, 1934.
2. Choisser, R. M., and Bloedorn, W. A. Agenesis of the lung. *Internat. Clin.*, 1939, *2*, 243–252.
3. De Weese, E. R., and Howard, J. C., Jr. Congenital absence of lung diagnosed before death. *Radiology*, 1944, *42*, 389–391.
4. Elward, J. F. Roentgen aspects of congenital aplasia of lung. *Radiology*, 1936, *27*, 667–671.
5. Heerup, L. Case of absence of left lung. *Hospitalstid.*, 1927, *70*, 1165–1176.
6. Hurwitz, S., and Stephens, H. B. Agenesis of lung; review of literature and report of case. *Am. J. M. Sc.*, 1937, *193*, 81–87.
7. Jamuni, A., and Ellis, A. G. Note on case of congenital absence of left lung. *Am. J. M. Sc.*, 1938, *196*, 824–826.
8. Madigan, D. G. Case for discussion, with special allusion to congenital absence of lung. *Tubercle*, 1941, *22*, 144–153.
9. Stokes, H. L., and Brown, C. J. O. Agenesis of lung; report of case. *Med. J. Australia*, 1940, *1*, 49–50.
10. Van Loon, E. L., and Diamond, S. Congenital absence of right lung; its occurrence in healthy child. *Am. J. Dis. Child.*, 1941, *62*, 584–589.
11. Yampolsky, J., and Fowler, C. D. Agenesis of lung; report of case. *Arch. Pediat.*, 1938, *55*, 293–296.

ACUTE OBSTRUCTION OF THE COLON

DIFFERENTIAL DIAGNOSIS BETWEEN VOLVULUS AND CANCER OF THE SIGMOID COLON BY PRELIMINARY ROENTGENOGRAM*

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LESIONS of the sigmoid colon may first make their presence known by symptoms of an acute obstruction. The most common lesions that are apt to produce such an obstruction are cancer of the sigmoid and volvulus.

It is the purpose of this paper to present roentgenologic diagnostic criteria for the differentiation of the obstruction due to a cancer, from that obstruction due to a volvulus, which may be seen in a preliminary roentgenogram of the patient. The difference in the appearance of the bowel in the two conditions is due to the fact that one is the result of a slow-occurring obstruction, allowing the bowel to accommodate itself to the increasing pressure, while the other is due to a sudden obstruction.

A search through the literature shows that volvulus has been diagnosed from the roentgenogram by other observers. Høyer[5] mentions the roentgen findings in a volvulus of the sigmoid as a large loop of distended bowel rising out of the pelvis. These are similar to the findings in the cases reported in this paper.

Miller,[11] reporting a case of volvulus in a child, mentions a large loop of gas distended bowel in the roentgenogram taken preoperatively, but fails to state whether the diagnosis of volvulus of the sigmoid was made from this roentgen finding. Volvulus has been diagnosed roentgenologically by the use of the barium enema (Hall[2]). None of the above mentioned authors point out differential criteria for a roentgen diagnosis of the slowly growing obstruction of the sigmoid which may be present in the cancer, from the sudden obstruction by a twisting of the bowel which is present in the volvulus.

Volvulus is a twist in the bowel usually over 180° and may occur anywhere in the gastrointestinal tract. It accounts for about 10 per cent of all cases of bowel obstruction (Wangensteen[13]). The sigmoid colon is especially likely to become twisted if it has a long mesentery with a narrow base in proportion to its length. There is a great variation in the normal length of the sigmoid, and the longer the loop and the closer its fixed ends, the greater the likelihood of volvulus. Chronic volvulus can occur (Holmgren[4]) with few symptoms, but it is the acute volvulus with which we are concerned since this is the case which requires prompt diagnosis and proper treatment or the mortality will be high.

Cancer of the sigmoid and volvulus of the sigmoid may make their presence known for the first time by a sudden bowel obstruction. Both types of cases will have a distended abdomen, some pain, but may have a normal pulse, temperature and white count. A correct diagnosis is imperative before any operative procedure is instituted. In cancer of the sigmoid, a right-sided incision for a cecostomy is in order to relieve the distention, and to drain the bowel. It is also advantageous to have the stoma as far as possible from the subsequent left side incision for a resection. On the other hand, a right-sided incision is of no value for a sigmoid volvulus. Here the choice is a left paramedian incision. Hence the necessity for a differential diagnosis.

It has been a routine procedure in our hospital[1,6,7,8,9] to take preliminary roentgenograms of all abdominal cases where an obstruction is suspected. It has been possible to make a differential diagnosis on the preliminary roentgenogram between a

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cancer in the sigmoid from a volvulus of the sigmoid in a few instances when such cases presented themselves.

A review of the course of events in both these conditions will suggest the criteria that may be available in both instances. Both depend upon gas appearing in the bowel. Gas may normally be present in the colon, but the colon is under no undue distention. With a block from either cause there is an accumulation of gas. It is the character of the gas-filled loops that differentiates the two conditions.

Cancer of the Sigmoid Colon. This is usually a slowly growing tumor within the lumen of the bowel. This part of the colon is narrow and muscular. With increasing obstruction it maintains its tone and size. On the contrary, the cecum and ascending colon, being of greater diameter and thinner of wall, acccommodate themselves to the increasing back pressure by dilating. The dilatation of the cecum may progress to such an extent that rupture occurs before symptoms of sigmoid obstruction appear, and the patient may be seen first with right-sided peritonitis (Saeltzer and Rhodes[12]). Most often the patient is rushed to the hospital, with the abdomen markedly distended and in pain, considered to be suffering from acute intestinal obstruction without cancer being suspected.

A roentgenogram of the abdomen in the supine or prone position will show a colon filled with gas, to the point of obstruction. The cecum and ascending colon are on the right side, they are dilated, lie in a vertical direction, and may have redundant loops. The transverse colon crosses the abdomen, it may also have redundant loops and is also dilated. The descending colon is well seen on the left side lying in a vertical direction, either not distended or with relatively little distention. The interruption of the gas-filled bowel is seen to be sharp at the point of obstruction. No large gas-filled loops are seen rising out of the pelvis.

Cancer obstructing other parts of the colon causes a similar appearance, i.e. gas-distended colon extends to the point of obstruction. In all instances careful observation of the appearance of the gas-distended loops will show it to be colon which shows a continuity of distention to the point of obstruction. If the ileocecal valve is competent the distention is limited to the colon, but it has been observed that the small intestine has been distended by obstruction of the colon.

Volvulus of the Sigmoid. In this instance there is a sudden twisting of the sigmoid bowel loop and it becomes a closed loop obstruction in relation to the rest of the bowel. This closed loop rapidly becomes greatly distended with gas and is seen to rise out of the pelvis and occupy the middle of the abdomen. It may extend to the diaphragm. At the summit the loop makes a sharp hair-pin turn. The origin of the gas is questionable. Weinstein[14] explains the accumulation of the gas as the result of the torsion completely cutting off the circulation, causing a deficient arterial oxygen supply to the involved loop. As there is some circulation to the bowel, with no impairment of the venous flow, an exchange of gases between the impaired circulation and the gas in the bowel takes place, carbon dioxide replacing the oxygen in the involved loop. The increasing amount of carbon dioxide plus other gases makes an ideal culture medium for the anaerobic organisms normally present in the colon with subsequent development of more gas. To substantiate this view he cites the foul odor that is present in a volvulus due to the anaerobic gases developed.

If a torsion is complete, the fluid in the bowel will exceed the gas present (Wangensteen[13]). A strangulated, gangrenous bowel may show no gaseous distention, either of the involved loop or of the bowel proximal to the volvulus.

It is of aid to take anteroposterior or posteroanterior roentgenograms with the patient lying on his side. Often a small amount of gas present with considerable fluid in the obstructed gut can be demonstrated, when no gas can be seen in the routine supine position.

The volvulus itself may act as a mechanical block, resulting in dilatation of the bowel proximal to it. This distended bowel never reaches the dilatation of the twisted loop and by its position can be identified as colon.

If the twist is complete and the acute obstruction is not promptly relieved, the bowel wall will become edematous, followed by gangrene, perforation and peritonitis.

Thus, an early differential diagnosis between the two types of obstruction is most important, so that the proper surgical procedure can be instituted.

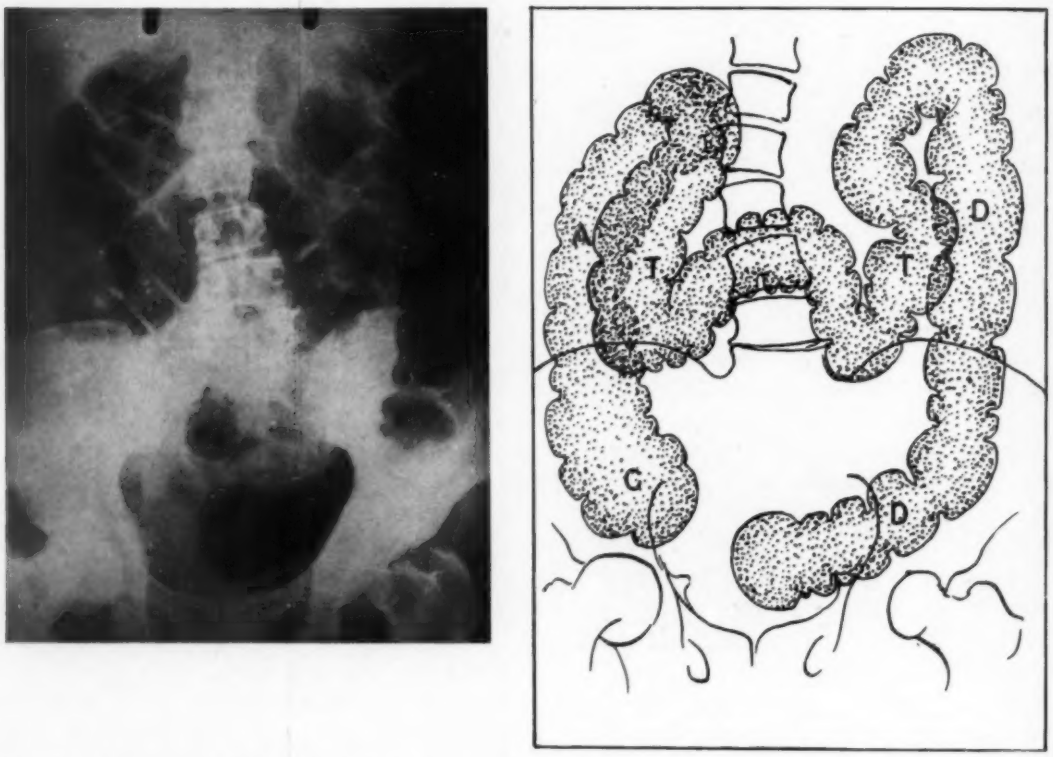

FIG. 1. Case I. Cancer of sigmoid colon. Colon distended with gas to the sigmoid. Redundant loops of transverse colon. Diagnosis: Mechanical obstruction of the sigmoid, probably due to cancer. *C*, cecum; *A*, ascending colon; *T*, transverse colon; *D*, descending colon.

CASE REPORTS

CASE I. B. G., female, aged fifty-eight, had an onset of cramp-like pains in the left lower quadrant at regular intervals of three to five minutes eighteen hours pior to entry. With the onset of pain the abdomen became distended.

On admission to the hospital, examination showed a distended abdomen, tympanitic but soft. No masses could be palpated.

The temperature, pulse and respiration were normal. The white blood count was 8,500, with a normal differential; red blood count was 3,560,000.

Roentgen examination of the abdomen showed a distended colon to the middle of the sigmoid (Fig. 1). A diagnosis was made of a mechanical obstruction of the sigmoid colon, probably due to cancer.

A first stage cecostomy was done, followed in a few weeks by the removal of a carcinoma. There was insufficient bowel to perform an anastomosis so a colostomy was done.

CASE II. E. S., female, aged sixty-eight, enjoyed good health until twelve hours before admission to the hospital. At that time she complained of griping pain in the left lower quadrant increasing in severity.

Examination on admission to the hospital showed a moderately distended abdomen; no palpable masses. The temperature, pulse and respiration were normal.

Roentgen examination of the abdomen showed a distended colon with redundant loops of transverse colon. The distention extended down the descending colon to the sigmoid (Fig. 2). Diagnosis was made of mechanical obstruction of the descending colon, probably due to cancer.

Roentgen examination of the abdomen in supine position showed a distention of the colon extending to the rectosigmoid (Fig. 3). A diagnosis was made of mechanical obstruction of the rectosigmoid, probably due to cancer. This was confirmed by a barium enema where an obstruction was met at this point.

A cecostomy was performed followed in two weeks by a resection for cancer and a permanent colostomy.

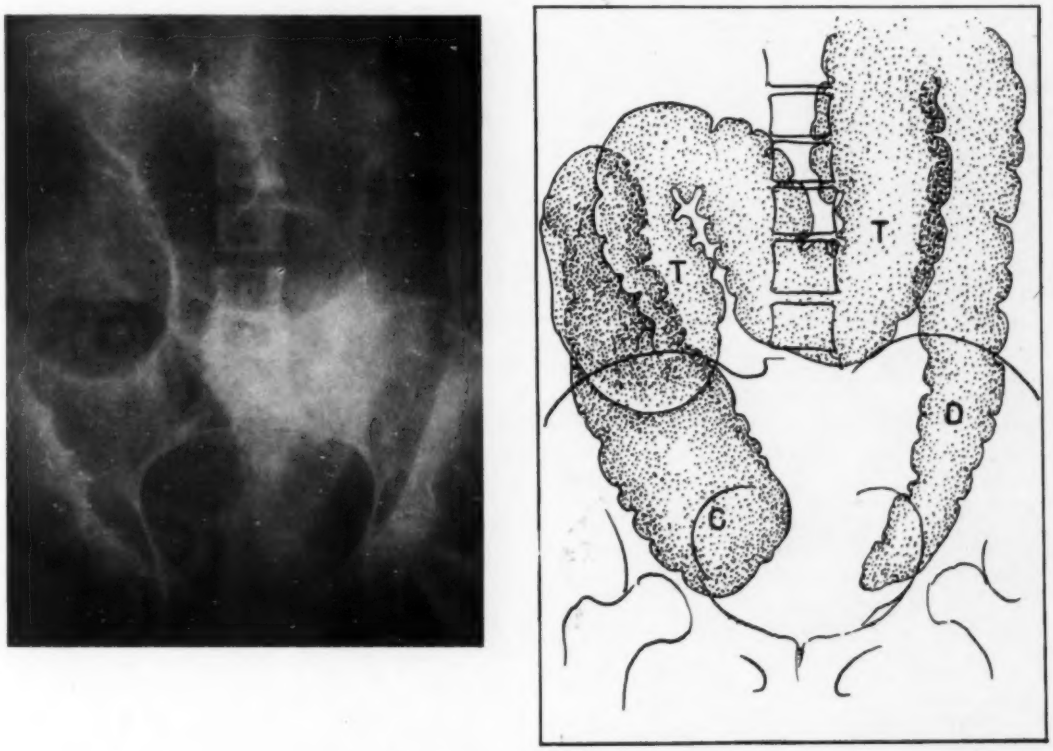

Fig. 2. Case II. Cancer of sigmoid colon. Colon distended with gas to the sigmoid. Redundant loops of descending colon. Diagnosis: Mechanical obstruction of the sigmoid, probably due to cancer. *C*, cecum; *A*, ascending colon; *T*, transverse colon; *D*, descending colon.

A first stage cecostomy was done followed in a few weeks by a resection for cancer and an end to end anastomosis.

Case III. B. S., female, aged sixty-six, had intermittent cramps in the left lower quadrant and a sense of fullness on the right side for two days prior to admission to the hospital.

Examination in the hospital showed a distention of the abdomen with no localized tenderness. Temperature, pulse and respiration were normal. White and red blood counts were normal.

Case IV. C. M., male, aged fifty, had a sudden onset of griping abdominal pain and distention following his evening meal. The pains were intermittent and mostly in the lower quadrants. He had daily bowel movements and vomited only once.

He entered the hospital three days after the onset. Examination showed a moderately distended abdomen, no tenderness or masses. The temperature, pulse and respiration were normal. The white blood count was 10,350, with normal differential, and the red blood count was 4,540,000.

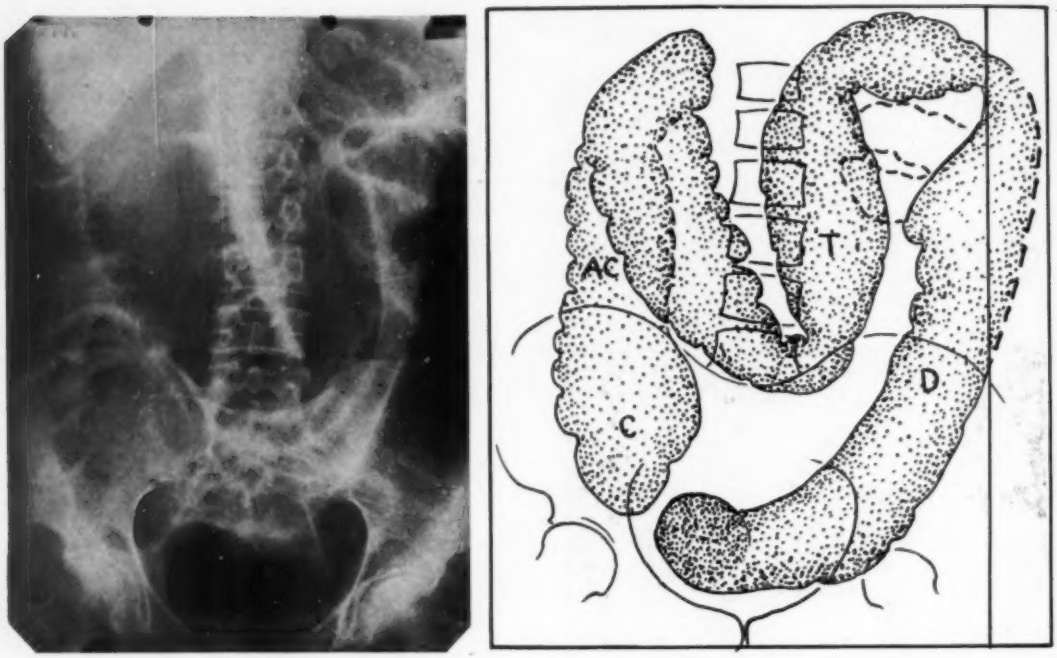

FIG. 3. Case III. Cancer of sigmoid colon. Colon distended with gas to the junction of the descending colon and sigmoid. Redundant loops of transverse colon. Diagnosis: Mechanical obstruction at junction of descending colon with sigmoid, probably due to cancer. *C*, cecum; *T*, transverse colon; *D*, descending colon.

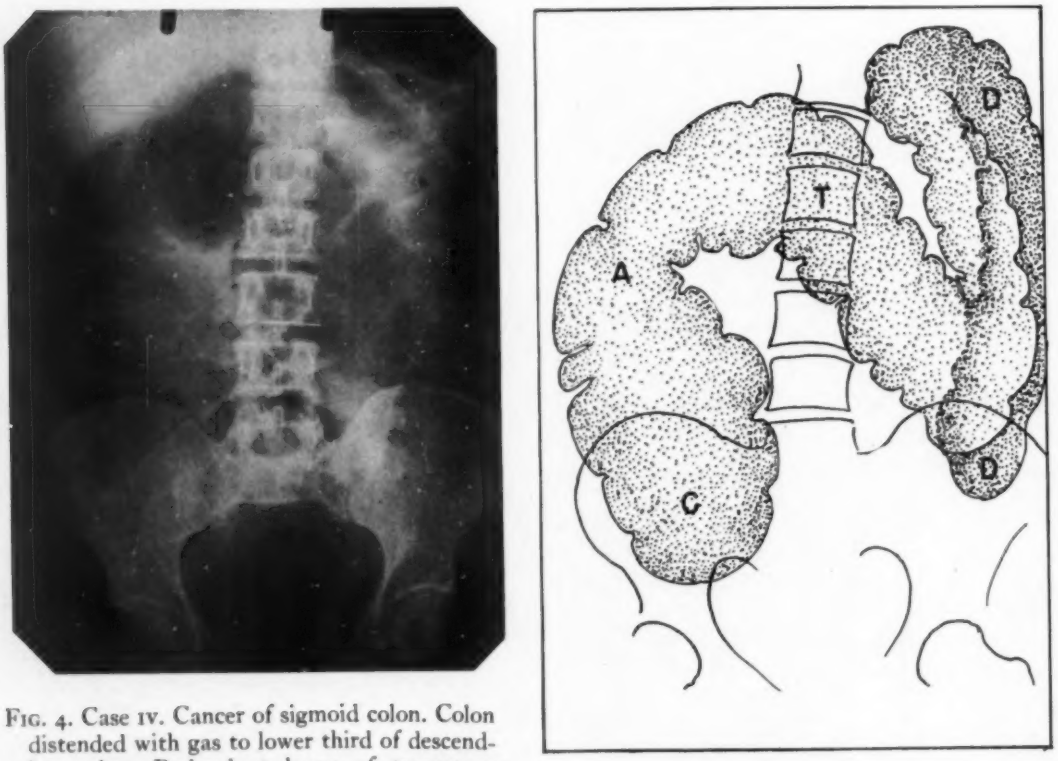

FIG. 4. Case IV. Cancer of sigmoid colon. Colon distended with gas to lower third of descending colon. Redundant loops of transverse colon. Diagnosis: Mechanical obstruction of descending colon, probably due to cancer. *C*, cecum; *A*, ascending colon; *T*, transverse colon; *D*, descending colon.

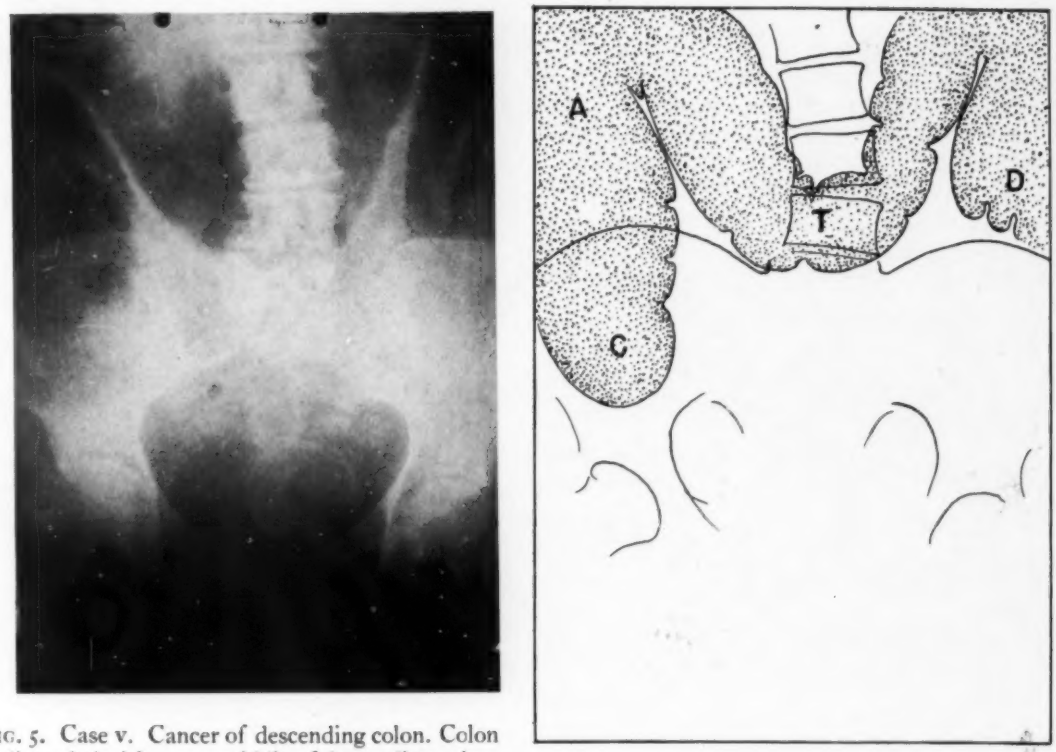

Fig. 5. Case v. Cancer of descending colon. Colon distended with gas to middle of descending colon. Diagnosis: Mechanical obstruction in the middle third of descending colon, probably due to cancer. *C*, cecum; *A*, ascending colon; *T*, transverse colon; *D*, descending colon.

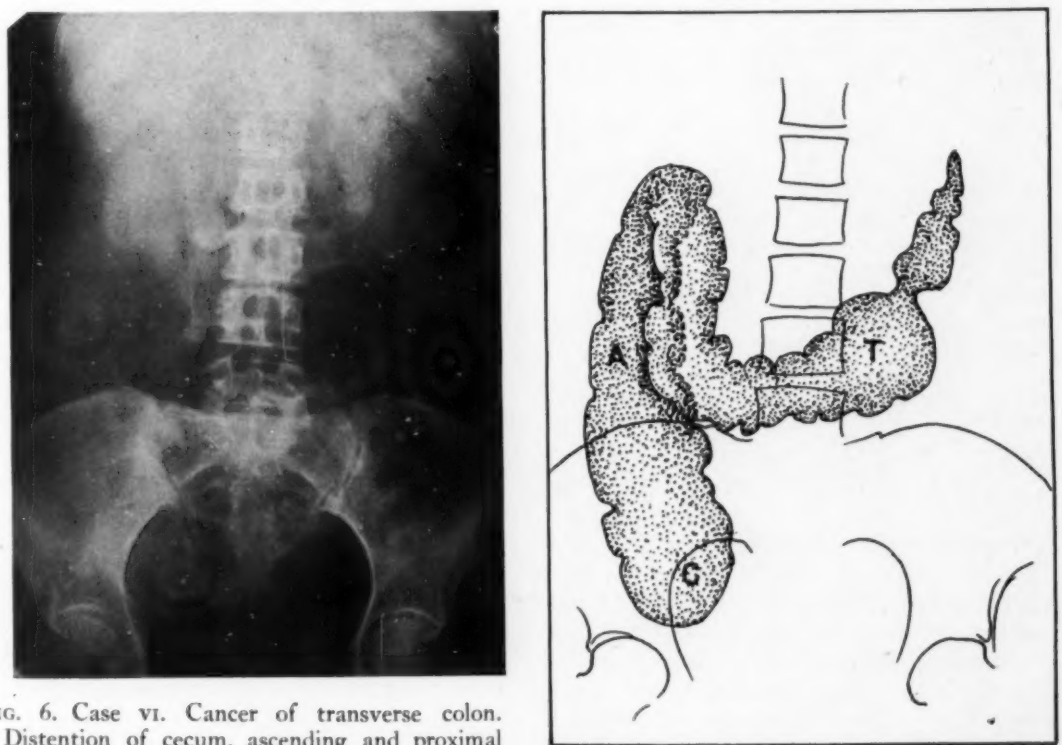

Fig. 6. Case vi. Cancer of transverse colon. Distention of cecum, ascending and proximal half of transverse colon. The transverse colon gradually tapers off, becomes funnel-shaped and finally becomes occluded. No gas is seen in bowel distal to this point. Diagnosis: Mechanical obstruction in middle third of transverse colon, probably due to cancer. *C*, cecum; *A*, ascending colon; *T*, transverse colon.

Roentgen examination of the abdomen in supine position showed distention of the colon to the sigmoid where the gas-filled colon abruptly stopped. The cecum was markedly distended (Fig. 4). The diagnosis was mechanical obstruction of the sigmoid at the junction with the descending colon, probably due to cancer.

A first stage cecostomy was done followed three weeks later by a resection for cancer of the sigmoid and an end to end anastomosis.

CASE V. E. M., female, aged seventy-eight, was unable to pass feces or flatus for the past five days even with the aid of cathartics. The distention increased and the patient vomited on the fourth and fifth day. With the distention there were griping abdominal pains.

Examination in the hospital showed a greatly distended abdomen, without tenderness or rigidity. No peristaltic waves were audible. The temperature, pulse and respiration were normal. The red and white blood counts were normal.

Roentgen examination showed a colon distended from the descending colon at the pelvic brim to the cecum. The cecum was markedly distended (Fig. 5). Diagnosis was made of cancer of the descending colon at the head of the pelvic brim.

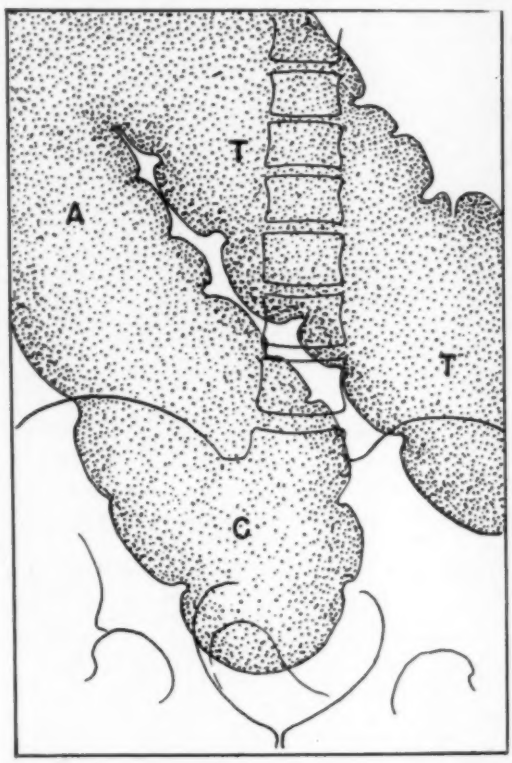

FIG. 7. Case VII. Cancer of transverse colon. The cecum, ascending and transverse colon are markedly distended, the distended transverse colon extending farther than shown on the roentgenogram. The diagnosis of an obstructed colon was unsuspected and the patient was sent for intravenous pyelography to determine the nature of a large right-sided mass. The left kidney pelvis is visualized and some dye is seen in the bladder. The diagnosis of an obstructed colon was obvious from the routine pyelograms. Diagnosis: Mechanical obstruction of transverse colon, probably due to cancer. *C*, cecum; *A*, ascending colon; *T*, transverse colon.

The patient's condition was poor, there was an auricular fibrillation, hypostatic pneumonia soon set in and she died.

The diagnosis of cancer was confirmed at autopsy.

CASE VI. The patient, female, aged fifty-two, had a sudden severe mid-abdominal pain, increasing in severity, which continued for three days before admission to the hospital. She had had no bowel movements during this time.

Examination of the abdomen showed no unusual distention. No masses were palpated. The

temperature was 36.6° C., pulse 70, red blood count 4,290,000 and white blood count 10,000.

Roentgen examination of the abdomen showed a distended colon proximal to the distal third of the transverse colon. At this point the colon could be faintly followed as a narrow, funnel-shaped, gas-filled lumen gradually narrowing until finally occluded (Fig. 6).

A diagnosis was made of an annular constricting carcinoma obstructing the transverse colon.

At operation an adenocarcinoma was found in the mid-portion of the colon. The lesion was removed and a Mikulicz type of operation and anastomosis performed.

Case VII. J. Y., male, aged seventy-two, had episodes of abdominal distress for the past two years. He never had pain, but would vomit and have three or four light watery stools a day, following which he would feel well for a week or more. The food vomited would be that eaten two to three days before. During the past two years he had lost 30 pounds.

Prior to admission to the hospital he had a gastrointestinal roentgen examination limited to the stomach, which was found to be normal. Examination in the hospital showed a dome-shaped abdomen, which was tense with a fullness present in the right side of the abdomen

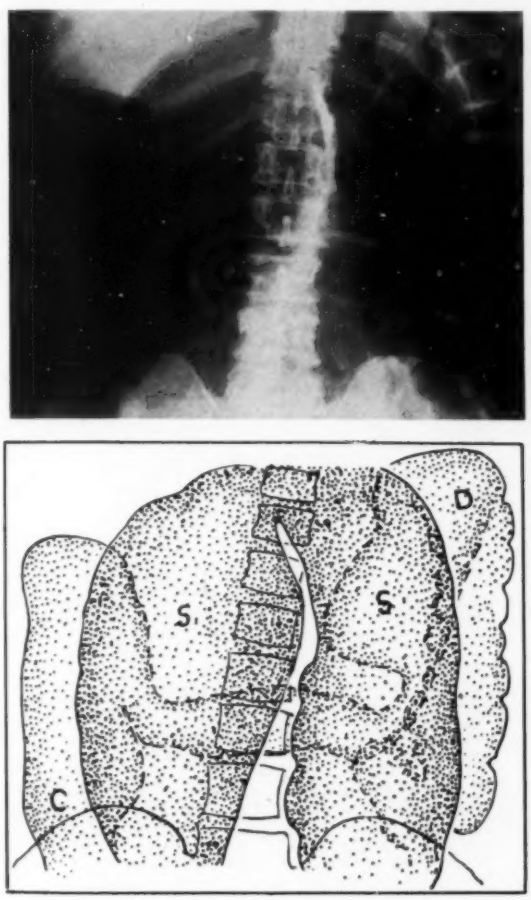

Fig. 8

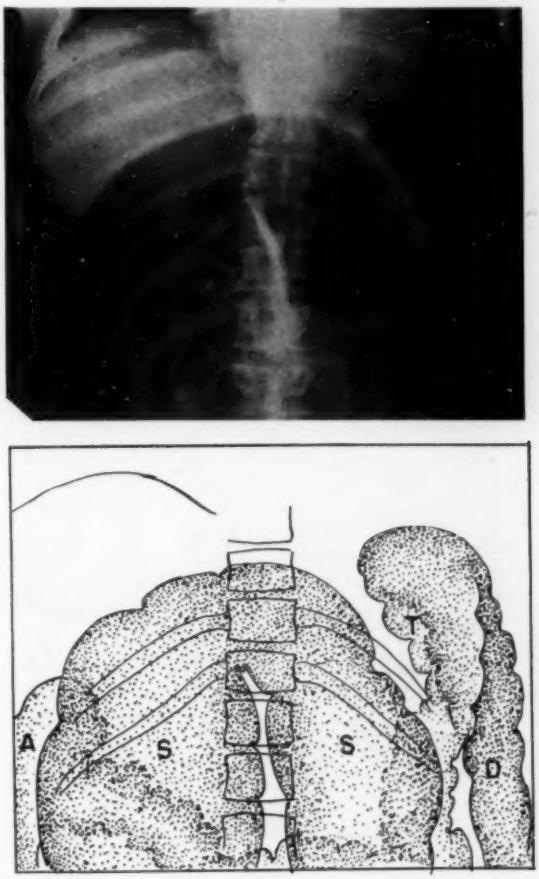

Fig. 9

Fig. 8 and 9. Case VIII. Volvulus of the sigmoid. The distended colon was so large that the entire bowel could not be demonstrated on one film. Figure 8, which missed the pelvis, shows a large loop of distended bowel rising out of the pelvis in the middle of the abdomen, extending towards the diaphragm. Moderately distended colon is seen behind the distended sigmoid loop. Figure 9 shows the upper end of this sigmoid loop with the sharp angulation between the ascending and descending limb. Diagnosis: Volvulus of sigmoid loop of colon with secondary distention of the colon proximal to the twist. *A*, ascending colon; *C*, cecum; *D*, descending colon; *S*, sigmoid.

To eliminate the possibility of a kidney tumor intravenous pyelography was done for which he entered the hospital. The temperature was 36.2° C., pulse 90. Blood count was normal.

The intravenous pyelogram demonstrated normal kidneys but they showed a markedly distended cecum, ascending and transverse colon (Fig. 7). The cecum had some barium from the previous gastrointestinal examination. No gas was seen in the colon distal to the obstruction. A diagnosis was made of an obstructive lesion in the transverse colon, probably cancer.

A preliminary cecostomy was done, followed later by removal of an annular carcinoma 2 inches wide encircling the transverse colon.

CASE VIII. C. W., male, aged sixty-nine, had low abdominal pain for the past six days associated with constipation. Repeated cathartics of cascara, castor oil, and salts had no effect. For the past two days the pain was more severe and the abdomen showed increasing distention.

Upon admission to the hospital the patient showed a markedly distended abdomen, which was tympanitic, no palpable mass. Rectal examination was negative. Temperature was 37° C., pulse 82, respiration 20.

Roentgen examination of the abdomen showed a markedly distended loop of bowel rising out of the pelvis, extending to the diaphragm. Two films were necessary to take in the whole abdomen (Fig. 8 and 9). The distended loop was interpreted as a loop of sigmoid and a diagnosis was made of a volvulus of the sigmoid.

At operation free fluid was found in the abdomen. The diagnosis of a sigmoid volvulus

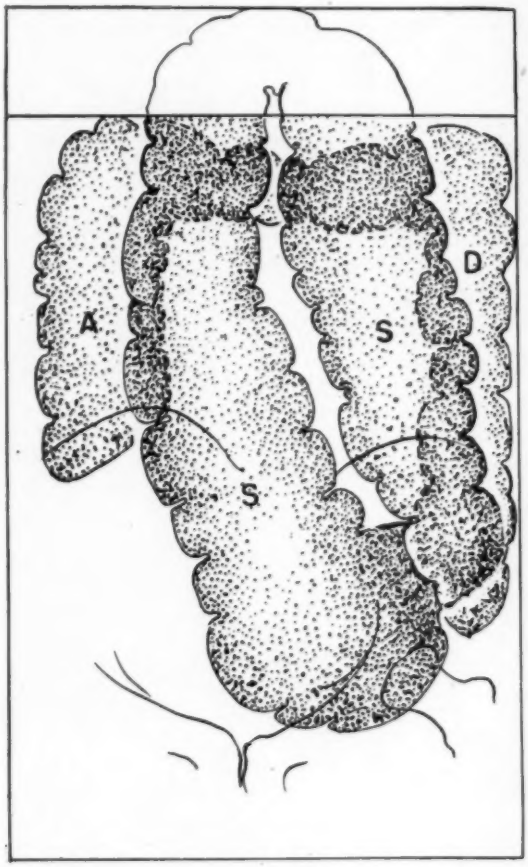

FIG. 10. Case IX. Volvulus of sigmoid. Tremendously dilated loop of bowel rising out of the pelvis and extending upward in the middle of the abdomen towards the diaphragm. The upper end of the bowel was cut off on the film and is diagrammatically presented. The colon proximal to this distended loop is also dilated, but not to the same degree. Diagnosis: Volvulus of the sigmoid. *A*, ascending colon; *D*, descending colon; *S*, sigmoid.

was confirmed. The bowel was drained, returned to the abdomen, but it resumed the old position of volvulus. The mesentery of the sigmoid was abnormally long. A colostomy was performed and subsequently the volvulus was reduced.

Case ix. C. L., female, aged fifty-seven, had a previous history of carcinoma of the breast with multiple bone metastases. She had had increasing abdominal pain for the past three days, associated with a marked distention of the abdomen associated with constipation.

On admission to the hospital the abdomen was markedly distended. Temperature, pulse and respiration were normal. Rectal examination was normal. The red blood count was 3,000,000; white blood count, 10,000.

Roentgen examination of the abdomen showed a markedly distended loop of bowel rising out of the pelvis which was diagnosed as a volvulus of the sigmoid. The colon proximal to the volvulus was also distended (Fig. 10).

At operation the diagnosis was confirmed, the bowel was untwisted and a temporary colostomy performed.

SUMMARY AND CONCLUSIONS

1. Differential diagnosis between a slowly growing mechanical obstruction of the colon and an obstruction due to a sudden twist of the bowel as a volvulus can be made on a preliminary roentgenogram of the abdomen.

2. The slowly growing obstruction of the bowel is usually an annular carcinoma, non-fungating, cicatrizing in type. The bowel accommodates itself to the increasing obstruction by distending first in its thinnest part. This is the cecum. The entire bowel gradually becomes distended to the point of obstruction. The various parts of the colon can be delineated by the contrast of the gas in the preliminary roentgenogram. When the obstruction is complete the patient may present himself for the first time with symptoms of a lesion which has the nature of an acute obstruction of the bowel.

3. Volvulus of the sigmoid is a sudden obstruction. The involved loop of sigmoid becomes markedly distended. The distended sigmoid loop rises out of the pelvis and lies in the mid-abdomen. It may extend to the diaphragm. It can be recognized as such on the preliminary roentgenogram.

4. Differential diagnosis between mechanical obstruction of the sigmoid and volvulus of the sigmoid is important since the surgical approach to the two conditions is different.

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REFERENCES

1. Brunn, H., and Levitin, J. Roentgenological study of intestinal obstruction. *Surg., Gynec. & Obst.*, 1940, *70*, 914–921.
2. Hall, M. R. Roentgenological diagnosis of volvulus of sigmoid megacolon, with report of two cases. Am. J. Roentgenol. & Rad. Therapy, 1938, *39*, 905–927.
3. Hinton, D., and Steiner, C. A. Recurrent volvulus of sigmoid colon. *Ann. Surg.*, 1942, *116*, 147–149.
4. Holmgren, B. S. Ein Fall von 360° Sigmoideumvolvulus ohne Strangulation und mit unbedeutenden Beschwerden. *Acta radiol.*, 1938, *19*, 230–238.
5. Høyer, A. Roentgen diagnosis of intestinal obstruction. *Acta radiol.*, 1938, *19*, 409–432.
6. Levitin, J. Intraperitoneal localized abscess; roentgenologic aid in diagnosis. *Radiology*, 1941, *37*, 205–207.
7. Levitin, J. Ileocolic intussusception; diagnosis by x-ray without contrast media. *Am. J. Surg.*, 1941, *54*, 494–498.
8. Levitin, J. Small bowel distention; differential diagnosis of mechanical obstruction and paralytic ileus by means of x-ray examination. *Brunn, Med.-Surg. Tributes*, 1942, pp. 253–268.
9. Levitin, J., and Trauner, L. M. Roentgenological study of postoperative abdomen. *Surg., Gynec. & Obst.*, 1942, *75*, 510–514.
10. Lippincott, S. W. Case of massive volvulus of sigmoid with unusual gut distension. *Canad. M. Ass. J.*, 1937, *37*, 489–490.
11. Miller, E. M. Gangrene of sigmoid flexure of colon due to volvulus. *Arch. Surg.*, 1940, *41*, 403–407.
12. Saeltzer, D. V., and Rhodes, G. K. Diastasic perforation of normal cecum resulting from obstruction of colon. *Ann. Surg.*, 1935, *101*, 1257–1263.
13. Wangensteen, O. H. The Therapeutic Problem in Bowel Obstructions. Charles C Thomas, Springfield, Illinois, 1937, Chap. xx, pp. 170–277.
14. Weinstein, M. Volvulus of cecum and ascending colon. *Ann. Surg.*, 1938, *107*, 248–259.

MECKEL'S DIVERTICULUM*

By M. E. MOTTRAM, M.D.,** *and* L. H. GARLAND, M.D.†

THE roentgen diagnosis of Meckel's diverticulum is rarely made preoperatively. The correct diagnosis of the anomaly is of considerable importance in cases of obscure abdominal pain and intestinal bleeding, especially since its surgical relief can often be fully assured. Additional cases correctly diagnosed prior to surgery would therefore seem to merit reporting.

The literature discloses reports of approximately 21 cases correctly diagnosed roentgenologically. Rousseau and Martin,[13] in 1943, collected 12 such cases‡ and added 1 of their own. In addition to these 13 cases, Conrad[4] and Ledoux[7] each reported 1 case; and Albright and Sprague[1] stated that, in a series of 22 cases of malignant disease involving Meckel's diverticulum, 6 cases had been demonstrated roentgenologically. These last mentioned authors did not publish any illustrations of their roentgenograms, but the other authors referred to did.

ANATOMICAL CONSIDERATIONS

Meckel's diverticulum is an unobliterated remnant of the vitelline duct, the omphalomesenteric structure which connects the alimentary canal with the umbilical vesicle in the embryo. This duct normally disappears by the seventh week of fetal life, but may persist in part or in toto. It is usually thought of as connecting with the terminal ileum, but embryologically there is no valid reason why, if persistent, it may not be found almost anywhere along the middle two-thirds of the alimentary tract. Surgical reports indicate that such wide variations in location do exist.

When the entire vitelline duct persists it may do so as a fibrous band or as a tubular structure (forming an intestinal fistula). When the distal end alone persists, an umbilical sinus occurs; a persistent mid-portion will form an enterocystoma; a persistent proximal portion will constitute a Meckel's diverticulum or sac. Of these various possibilities, the persistent proximal portion (or Meckel's diverticulum) is the most common.

The diverticula vary widely in size, shape, location and anatomical structure. The size may vary from a tiny pouch to an enormous sac. The "usual" size reported at autopsy in uncomplicated cases is from 1 to 3.5 cm. in diameter (Noel[9]). In cases complicated by tumor, the reported size is almost invariably larger.

Moll[8] reported a case 82 cm. in length, described as virtually a "reduplication of the bowel!" Goldstein and Craig reported one 66 cm. in length; Chaffin[3] one 95 cm. in length in an eight year old child; and Pollard[11] one 90 cm. in length.

The most common point of origin is from the small bowel, usually the ileum about 37 cm. (15 inches) proximal to the ileocecal valve. However, it is often found anywhere from 3 to 100 cm. above that valve and it is stated that it may even arise from the colon. We have gained the impression that the ordinary asymptomatic cases tend to occur close to the ileocecal valve, while those with surgical complications are more apt to be 50 to 60 cm. (20 to 24 inches) proximal thereto. This impression is strengthened by the fact that anatomists usually describe the lesion as arising from the terminal ileum, while surgeons often place it at about 50 cm. above the ileocecal valve. It usually lies free on the antimesenteric border of the bowel, but may vary in its point of origin, and even have its own mesentery. It may be considerably distorted in shape owing to the presence of adhesions, secondary infection or tumor.

The diverticulum may have walls iden-

‡ Including those of Case[2] and Farr and Penke.[6]

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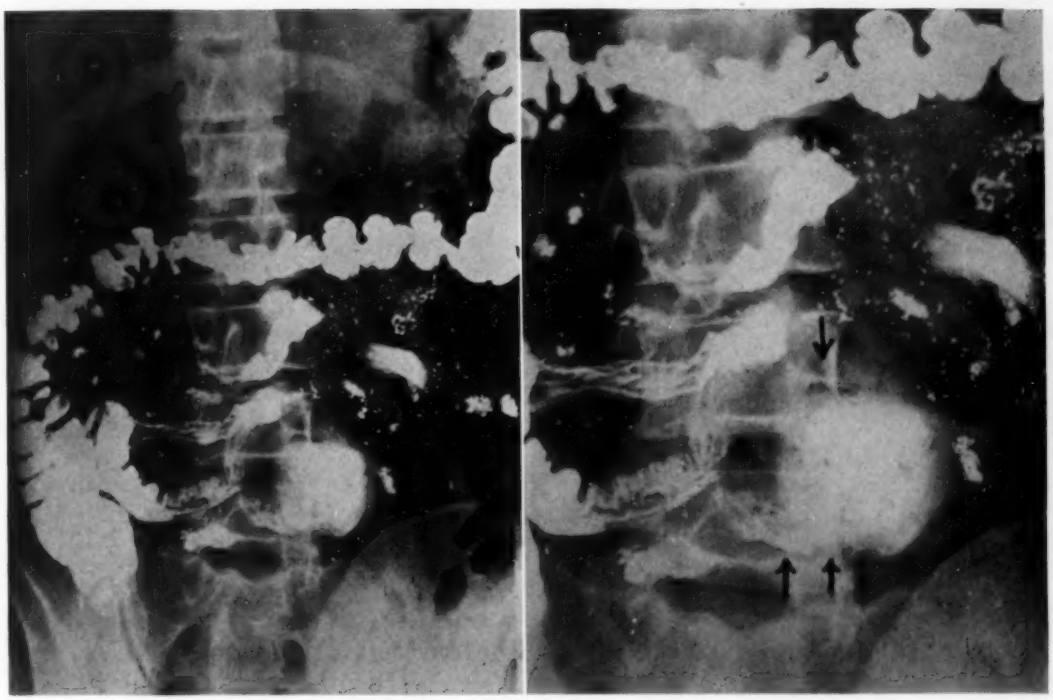

Fig. 1 Fig. 2

Fig. 1 and 2. Fig. 1. Meckel's diverticulum. Six hour roentgenogram at time of initial examination. Note collection of opaque material overlying the left side of the lower lumbar spine. Roentgenoscopic survey prior to ingestion of the barium disclosed no such opacity (thereby excluding a calcifying mesenteric cystic lesion or prior medicinal material in the bowel). Note that there is no significant stasis proximal to the diverticulum and that the adjacent loops of ileum are not dilated.

Fig. 2. Same as Figure 1, with arrows pointing to the margins of the sac and, at its inferomesial edge, the neck of the sac. The barium is obviously diluted with non-opqaue material, presumably present in the sac prior to the roentgen examination. Subsequent operation disclosed this sac to be about 7 cm. in diameter, located about 60 cm. proximal to the ileocecal valve, and partly filled with purulent material.

tical in structure with those of the adjacent intestine, or one of the muscle layers may be absent. Heterotopic gastric or duodenal glands may be present. Neoplastic alteration is not uncommon.

INCIDENCE

The incidence of Meckel's diverticulum in the general population, at autopsy, is about 2 per cent. Of those so affected, Wilson[15] estimated that about 20 per cent will develop symptoms or complications, leading to its diagnosis, usually at operation.

COMPLICATIONS

Normally, the majority of cases of Meckel's diverticulum are asymptomatic and uncomplicated. A small number develop intestinal obstruction (with or without intussusception[12]); diverticulitis (with or without perforation); ulceration (with perforation or hemorrhage); foreign body inclusion; or malignant degeneration.[14]

If the diverticulum has walls deficient in one or more of the muscle layers, stasis tends to occur. If gastric mucosa is present, peptic ulceration may develop, either in the diverticulum itself or in the adjacent ileum; secondary intestinal bleeding may then occur.

DIAGNOSIS

The diagnosis can often be suspected and occasionally made on clinical grounds alone. Symptoms of intermittent mid or lower abdominal pain, with or without

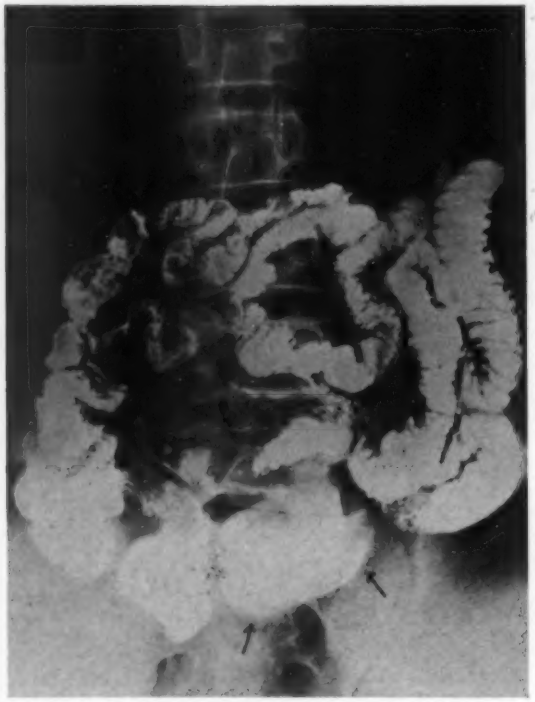

FIG. 3. Meckel's diverticulum. Roentgenogram made two and a half hours after oral ingestion of barium water. Note ovoid opacity (barium-filled diverticulum) overlying the lumbosacral angle.

tarry stools, may suggest diverticulitis or ulceration in a Meckel's sac, especially when occurring in individuals under thirty-five years of age. Intermittent intestinal bleeding without symptoms or signs of peptic ulcer, intestinal tumor or colonic disease may also lead to suspicion of the lesion.

Roentgen examination of the alimentary tract will only rarely establish the presence of the condition, but the recorded cases, including that reported herewith, demonstrate the fact that such diagnosis is occasionally possible. We doubt if the small (2.5 cm. diameter) diverticulum can be diagnosed, except fortuitously. The larger diverticula, especially those with abnormal or inflamed walls (and therefore with stasis) should be detected on careful gastrointestinal examination.

Gastrointestinal study with small water-barium meals (up to 8 ounces volume) will reveal a collection of barium in the mid or lower abdomen, which does not conform to the shape or appearance of the normal loops of small bowel. This collection of barium will vary in density during a twenty-four hour period according to its degree of dilution with intestinal contents. It may not be demonstrable at times, presumably owing to prior filling of the sac with intestinal contents. Pfahler[10] reported 1 case in which the diverticulum filled only in the prone position. Ehrenpreis[5] suggested that a lateral projection would aid in diagnosis, because of pointing of the diverticulum towards the umbilicus. This might aid in the case of a very large diverticulum, but the unavoidable overlapping of adjacent, filled, small bowel loops would render accurate delineation very difficult. Further, the marked variation in the direction of most diverticula would probably render the view of little or no value.

FIG. 4. Meckel's diverticulum. Roentgenogram made three and a half hours after oral barium ingestion. The sac is slightly altered in shape and location.

The following case showed an encapsulated collection of barium connected with the lower ileum and persisting for twenty-four hours:

G. W. C., a male Hawaiian aged thirty (referred by Dr. J. Tobin to Dr. M. E. Mottram for gastrointestinal study). The patient gave a history of steady epigastric and periumbilical pain, of several hours' duration. The pain came on suddenly and gradually extended to the lower abdomen. There was nausea but no vomiting. The patient was admitted to the hospital, the pain subsiding on entry, leaving generalized abdominal tenderness. Prior to this attack there had been milder bouts of abdominal pain and gaseous distention for a period of one year.

On examination in the hospital, the patient had periumbilical and left lower quadrant tenderness, with moderate muscle spasm, but no rebound tenderness. Peristalsis appeared to be decreased. His temperature was normal (but rose slightly next day to 99.8° F.). The white blood count was 18,150 with 87 per cent polymorphonuclear cells; next day it was 13,300, with 72 per cent polymorphonuclears. No stool examination was done.

Roentgen Examination. Preliminary roentgenoscopic survey of the abdomen was negative. The esophagus, stomach, and duodenum appeared normal. At six hours, a collection of barium was noted in the mid-abdominal area, apparently in a pocket or sac near the umbilicus. Re-examination next day confirmed the presence of this sac-like structure, which had then changed in size. It measured about 9 by 5 cm. in the posteroanterior projection, was poorly movable and slightly tender. It retained some barium for over twenty-four hours. There was no small bowel obstruction. The appendix filled, was movable and not tender. The colon appeared negative. A diagnosis of probable Meckel's diverticulum was made.

Surgical intervention was decided upon by Drs. Curtis Smith and J. Tobin. At operation a large, circular diverticulum, measuring about 7 cm. in diameter was found attached to the ileum about 60 cm. (24 inches) proximal to the cecum. Its neck was about 2.5 cm. in diameter and it had a short mesentery. Its external surface was red and granular, and some loops of small bowel were lightly adherent to its apex. The diverticulum was removed; it weighed 126 grams and contained intestinal contents, thick purulent material and barium. The stump was inverted and the abdomen closed. The patient made an uneventful recovery.

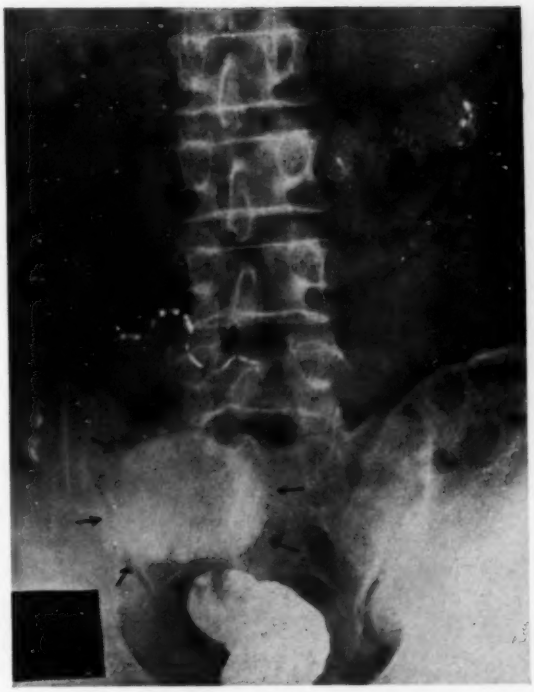

FIG. 5. Meckel's diverticulum. Roentgenogram made twenty-four hours after barium by mouth. The sac remains filled with diluted barium. The small bowel loops are empty. The appendix is filled and displaced slightly cephalad by the sac. A little barium remains in the colon, chiefly in the rectum.

SUMMARY

1. Meckel's diverticulum occurs in about 2 per cent of the population. It varies greatly in size, shape, and location. The larger diverticula apparently tend to produce symptoms, principally those of intestinal bleeding, attacks of colicky pain localized about the umbilicus, and vomiting.

2. The diverticulum frequently occurs at a greater distance proximal to the ileocecal valve than is thought probable (e.g. a distance of 60 cm. as in the case reported herewith).

3. The finding of a collection of barium in the mid or lower ileal area, which fails to conform to the usual pattern of the small bowel or which persists after the small bowel is empty should suggest the possibility of the lesion.

4. Most of the reported cases of Meckel's diverticulum diagnosed roentgenologically

have been of fairly large size, or have been suspected clinically and carefully searched for. Some of them have been associated with ulceration, obstruction or tumefaction.

5. The lack of uniformity in the location and size of the diverticulum, and the fact that at times it is filled with non-opaque material may render the roentgen diagnosis very difficult.

REFERENCES

1. ALBRIGHT, H. L., and SPRAGUE, J. S. Primary adenocarcinoma in Meckel's diverticulum. *New England J. Med.*, 1942, *226*, 142–146.
2. CASE, J. T. Jejuno-ileal diverticula. *Acta radiol.*, 1926, *6*, 230–240.
3. CHAFFIN, L. Surgical emergencies during childhood caused by Meckel's diverticulum. *Ann. Surg.*, 1941, *113*, 47–56.
4. CONRAD, H. A. Meckel's diverticulum. *Am. J. Surg.*, 1941, *52*, 267–274.
5. EHRENPREIS, B. Roentgen diagnosis of Meckel's diverticulum. AM. J. ROENTGENOL. & RAD. THERAPY, 1939, *42*, 280–284.
6. FARR, C. E., and PENKE, M. Meckel's diverticulum. *Ann. Surg.*, 1935, *101*, 1026–1041.
7. LEDOUX, A. C. Meckel's diverticulum. *Radiology*, 1942, *38*, 728–729.
8. MOLL, J. Quoted by Chaffin.[3]
9. NOEL, W. W. Meckel's diverticulum. *Am. J. Surg.*, 1940, *49*, 454–463.
10. PFAHLER, G. E. Roentgenological diagnosis of Meckel's diverticulum. *Surg., Gynec. & Obst.*, 1934, *59*, 929–934.
11. POLLARD. Quoted by Chaffin.[3]
12. ROTHMAN, M., and ZETENA, D. F. Meckel's diverticulum as cause of intestinal obstruction. *Am. J. Surg.*, 1943, *60*, 443–446.
13. ROUSSEAU, J. P., and MARTIN, A. G. M., III. Meckel's diverticulum; preoperative roentgen diagnosis. *Radiology*, 1943, *40*, 605–607.
14. SKINNER, I. C., and WALTERS, W. Leiomyosarcoma of Meckel's diverticulum, with roentgenologic demonstration of diverticulum. *Proc. Staff Meet., Mayo Clin.*, 1939, *14*, 102–107.
15. WILSON, H. Meckel's diverticulum. *Am. J. Surg.*, 1942, *56*, 614–618.

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PERIOSTEAL LESIONS IN SCURVY*

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THE important developments in the history of scurvy are the accurate comprehensive description of the cause, symptomatology, pathology and cure of the disease in adults by James Lind, the English naval surgeon, in 1747, and the recognition of infantile scurvy, formerly called acute rickets, by Thomas Barlow[1] and a group of English pediatricians toward the end of the nineteenth century. The disease had undoubtedly become more prevalent at that time from the use of antiseptic and artificial methods of infant feeding, and Barlow was able to report his findings in 39 fatal cases. He was impressed by the frequency and severity of the subperiosteal hemorrhages occurring usually in the long bones, but rarely also in the flat bones such as the scapula and in the bones of the cranial vault and the orbits. It was Barlow's conception that the subperiosteal hemorrhages were analogous to the hemorrhages seen in the gums and in the skin of adult scurvy and that they *caused* a disturbance in the growth process at the ends of the long bones leading to epiphyseal separation and displacement. Epiphyseal separation had been described by Lind[9] as follows: "All young persons under 18 had in some degree their epiphyses separated from the body of the bone," but the similarity to the epiphyseal separation of infantile scurvy was not recognized until much later. It is important to recall that Barlow's studies were made on fatal cases and were therefore concerned only with cases where the disease was very severe and far advanced. Apparently no attempts were made to study the development of scurvy experimentally in animals at that time. The early bone changes in infants were, however, studied a little later by means of the recently discovered roentgen ray, notably in the hands of Fraenkel[8] whose name has been given to the dense metaphyseal line which he emphasized as an important

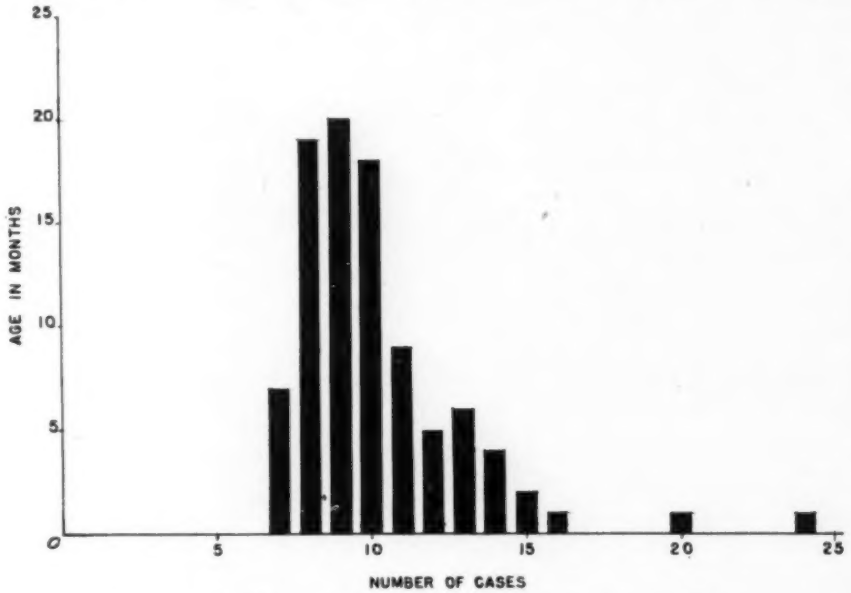

FIG. 1. Age incidence in 93 cases of infantile scurvy, 1936–1941.

* From the Department of Roentgenology of the Children's Hospital of Michigan.

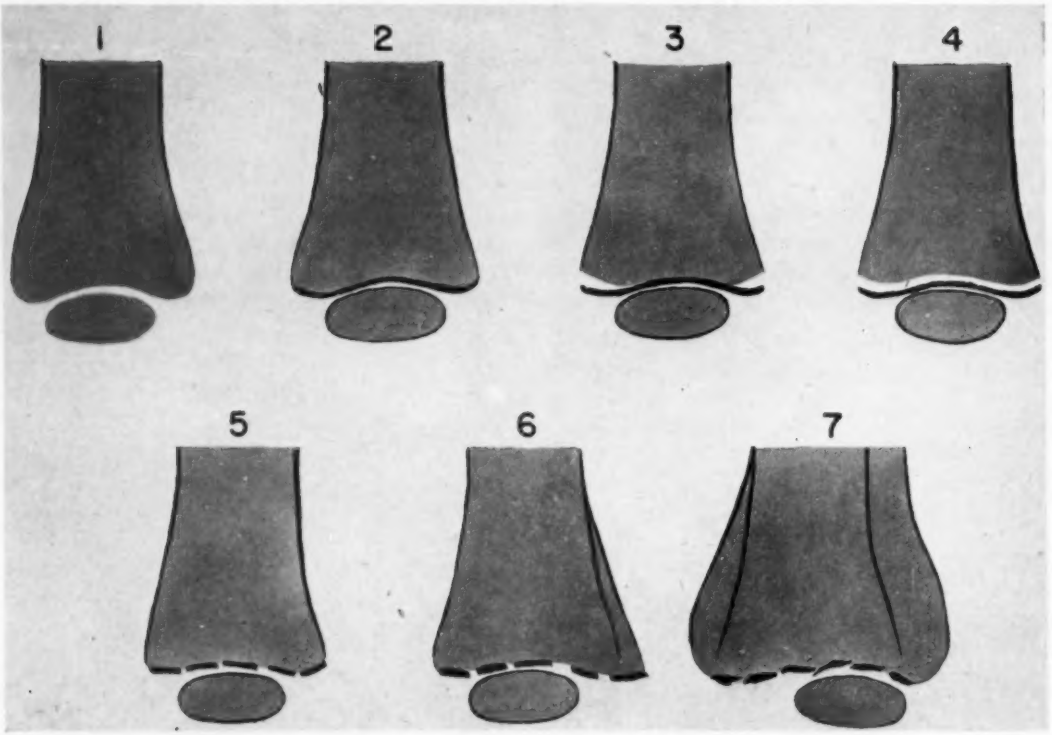

Fig. 2. A diagrammatic representation of the development of the osseous changes in scurvey. *1*, normal growing bone; *2*, increased width and density of the metaphysis, signet ring appearance of the epiphysis and a ground glass type of demineralization; *3*, submetaphyseal notching representing minute fractures of newly formed bony trabeculae; *4*, extension of the notching and separation of the metaphysis; *5*, fragmentation of the metaphysis; *6*, lateral displacement of a small metaphyseal fragment lifting the periosteum and causing subperiosteal hemorrhage Type I; *7*, further disintegration of the metaphysis permitting lateral displacement of the epiphysis and massive subperiosteal hemorrhage Type II.

sign of scurvy. Wimberger[15] saw a large number of cases in Vienna following the first World War and described the signet ring appearance of the epiphyses which has since been referred to by his name. By using refinements in roentgen technique and the correlation with animal experiments, the finer details of the metaphyseal changes in scurvy were worked out by Pelkan,[14] Bromer,[4] and Park *et al.*[13] It has gradually become evident that subperiosteal hemorrhages are only a late manifestation of scurvy and that the earliest osseous changes occur in the metaphyses. Although most close students of the subject agree on this point, the misconception is still prevalent that periosteal lesions are a prominent feature in the diagnosis and pathology of scurvy. Park and his associates in 1935 stated: "Subperiosteal hemorrhages develop only when the involvement of bone has reached an advanced stage. They are the result of the fractures of the cortex and lattice at the end of the shaft," and more recently in 1940: "The fractures of the lattice cause the characteristic extravasations of fibrin and sometimes hemorrhage." "The giving way of the lattice causes the cortex to break, the blood vessels of the periosteum to be torn, and blood to leak under the periosteum."

For the purpose of studying the correlation of the metaphyseal and periosteal lesions in scurvy as revealed roentgenographically, the roentgenograms were reviewed where a diagnosis of scurvy had been made at the Children's Hospital of Michigan during the years 1936–1941. Dur-

ing this six year period in a hospital service comprising 106,800 new out-patient visits and 41,773 hospital admissions, 93 cases were encountered indicating that even in these days of vitamin consciousness infantile scurvy is by no means a rare disease.

The sex distribution was about equal, comprising 48 males and 45 females.

The age incidence revealed a pronounced limitation of cases to the latter half of the first year of life. No cases were observed before the seventh month and 90 per cent fell in the ages from seven to thirteen months inclusive (Fig. 1). This corresponds well with the age incidence observed by McLean and McIntosh.[10]

All of the cases were tabulated with designations to indicate the presence and extent of the various osseous manifestations of scurvy revealed in the roentgenograms. The roentgenograms routinely comprised single anteroposterior views of both arms and legs. Examinations were often repeated a few weeks following the institution of treatment to follow the progress of the lesions. The most advanced lesions were usually seen in the knees, followed by the shoulder and ankle joints, and then by the wrist, hips and elbow joints in that order. The progression of lesions as the disease becomes more severe is represented schematically in Figure 2.

The earliest changes represented by the ground glass type of demineralization of the bone structure and by the signet ring appearance of the epiphyses are seen with

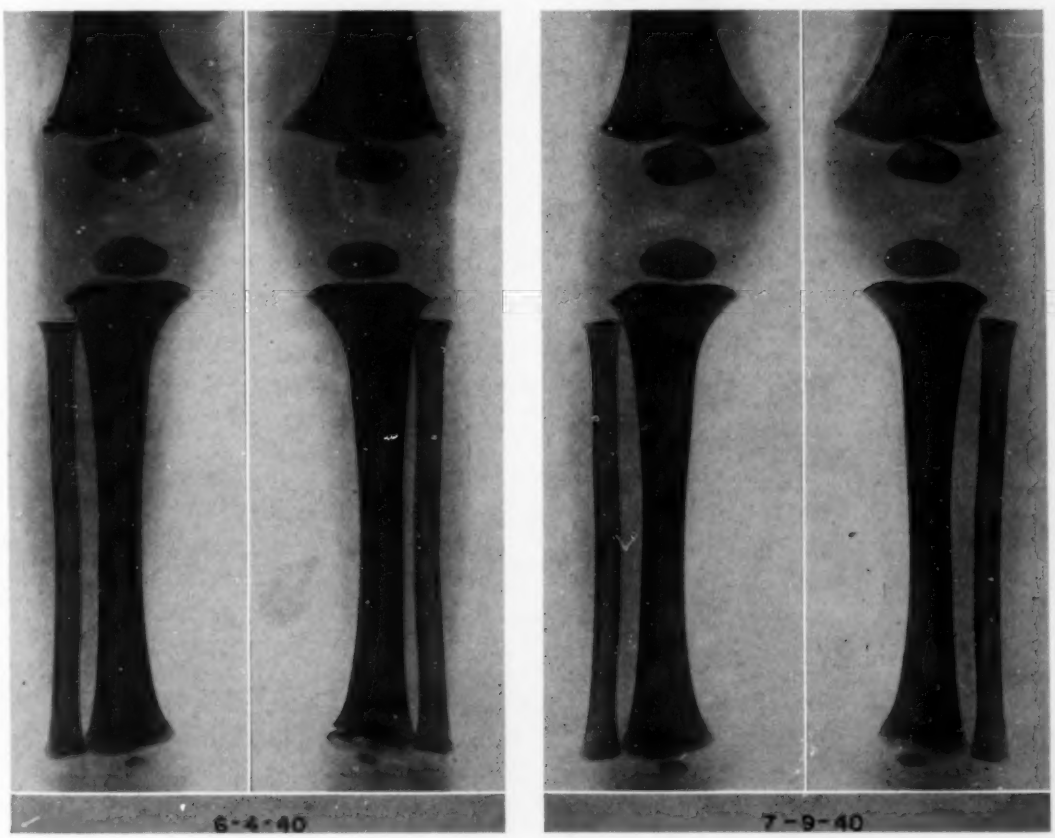

Fig. 3. Case 1. E. L. (CHM No. F3803). Well marked clinical and roentgen evidence of scurvy in a male infant, aged eight months. The metaphyseal lines are broad and dense, and there is well defined submetaphyseal notching especially in the lower ends of the femurs, but no metaphyseal fragmentation or displacement. Re-examination five weeks later following administration of cevitamic acid shows complete healing with no sign of a periosteal hemorrhage.

Fig. 4. Case II. L. O. (CHM Nc. G6913). A nine month old white female fed by formula and without orange juice or other antiscorbutic. Metaphyseal changes in the tibiae are minimal and no periosteal shadows are seen. Well marked scorbutic changes are seen in the lower femoral metaphyses. On the right there is only slight lateral extrusion of the metaphyseal fragments and there are Type I periosteal shadows, while on the left the metaphyseal extrusion is more marked and there has been some lateral displacement of the epiphysis as well. Here there is the Type II subperiosteal hemorrhage.

great frequency but are not, we think, of sufficient specificity to be regarded as pathognomonic of scurvy. The signet ring appearance was most pronounced in the epiphyses at the knee joints and was present in the same degree in all of the cases. The ground glass appearance, on the other hand, was seen less constantly (74 per cent) and was absent particularly in those cases complicated by rickets.

The submetaphyseal notch, emphasized by Park and his colleagues as the earliest pathognomonic sign of scurvy in the roentgen examination, was observed in 89 per cent of the 93 cases. In other cases it was doubtful, in some instances being masked by rickets. The metaphysis was found fragmented or separated in 42 per cent of the cases, and there was actual displacement of the metaphysis or a large portion of it carrying with it the epiphysis in 19 per cent.

Particular attention was paid to the character of the periosteal shadows and their association with the metaphyseal changes. Periosteal shadows in scurvy were observed to be of two types. The first, referred to as Type I, appears as a narrow triangular shadow having its base at the metaphysis and extending from there for some distance along the shaft. This type was not seen when there was only the submetaphyseal notch but first when there was some fragmentation of the metaphysis and a slight lateral displacement of a small marginal fragment at the lateral or medial aspect. This type of periosteal shadow was observed in 39 per cent of the cases.

The second type of periosteal shadow (Type II) is very much larger, is usually club shaped and extends along the greater length of the shaft. It is quite constantly associated with displacement of a large portion of the metaphysis and the epiphysis as well. This circumstance was observed in 17 per cent of the cases.

Plausible explanations are available for a small discrepancy which exists between these actual observations and this hypothetical association of metaphyseal and

periosteal lesions. As Nelson, Doughty and Mitchell[12] and others have pointed out, the periosteal hemorrhages only become demonstrable on the roentgenogram when calcification begins to take place. Calcification does occur after a very short interval but there is nevertheless a period when the circumstances for a subperiosteal hemorrhage may be present and the hemorrhage not be demonstrable for want of calcification. This is not a common occurrence and is obviated if serial studies are made, as they were frequently in this review. When a large subperiosteal hemorrhage is present, the displacement of the epiphysis may not be evident in the plane in which the roentgen exposure was made. One of the most common sites of epiphyseal displacement is at the lower end of the femur. The displacement occurs lateralward and if the examination is made with the child on his back and the legs in external rotation and some abduction, lateral views will be obtained of the knees and the lateral displacement of the epiphysis will be missed. This position is the most comfortable for the scorbutic infant who resents manipulation and was commonly used in the early studies. Epiphyseal displacement may also be missed because it is not present at the moment the study is made. It is well known that when epiphyseal displacement has occurred the epiphyses are quite freely mobile, and it is readily understandable that the epiphysis may by chance be in a normal position when there has recently been a marked displacement and a large subperiosteal hemorrhage. This circumstance was observed in one case in this series.

Fig. 5. Case III. B. B. (CHM No. G3657). A ten month old white female infant fed by formula and canned orange juice. Cod-liver oil had been adequate. The most advanced scorbutic changes are seen in the lower ends of the femurs where there has been complete disintegration of the metaphyses with lateral displacement of the epiphyses and Type II periosteal shadows. Lesser changes are seen in the tibiae but with some medial extrusion of metaphyseal fragments and a Type I periosteal shadow at the lower end of the right tibia.

Differential Diagnosis of Periosteal Changes Manifested Roentgenographically in Infancy

The periosteal areas rival the metaphyseal regions in the variety and striking character of the changes which occur in infancy during the period of most rapid growth. The number of diseases with great variations in their prognosis and indications for treatment which are associated with periosteal manifestations calls for a close interpretation and analysis.

Rickets. It is often difficult to identify minor degrees of rickets and scurvy when both diseases are present together. Some degree of rickets was thought to be present in 36 of the 93 cases in this series. At times, lesions characteristic of scurvy may be seen in the metaphysis of one bone and those of

Fig. 6. Case iv. M. S. (CHM No. D527). A one year old white male infant fed by cow's milk with no cereals or vegetables and no cod-liver oil or orange juice. The first study shows disintegration of the lower femoral metaphysis especially at the lateral aspects with some lateral extrusion of metaphyseal fragments and a signet ring appearance of the femoral epiphysis, characteristic of scurvy. At the second study ten days later there has been displacement of the epiphysis and formation of a large subperiosteal hematoma Type ii.

rickets in another as in Case viii. Rachitic changes are apt to be most pronounced in the distal ulnar metaphysis, while the distal radial metaphysis seems to be more prone to the development of a scorbutic lesion. When the manifestations occur together, as at the knee, the metaphysis is wider and with a more frayed irregular structure of lower density than when scurvy is present alone. Also the submetaphyseal notch occurs at a greater distance from the epiphyseal line and is less sharply defined. The periosteal manifestations may also provide distinguishing features. While in scurvy the periosteal shadows originate in the metaphyseal areas, are usually widest there and tend to have a uniform density with a sharp outline, the periosteal shadows of rickets occur about the middle third of the shaft and are laminated. The periosteal lesions of rickets are most pronounced in the first six months of life and are much less frequently seen in the age period when scurvy is prone to occur.

Periosteal shadows of unknown etiology such as those reported as occurring especially in early infancy[11] resemble the periosteal shadows of rickets and are easily differentiated from scruvy. In our experience, minor degrees of this periosteal manifestation are seen frequently. The shadows disappear in the course of a few weeks by incorporation into the cortex and do not seem to be of any clinical or pathological significance.

Infection. In infancy the bones frequently become involved in a variety of infections with prominent periosteal as well as osseous lesions. The lesions of syphilis, tuberculosis, and pyogenic osteomyelitis in general offer no roentgenologic problem in differentiation from scurvy. Clinically, however, scurvy is occasionally mistaken for osteomyelitis and there seems to be at least one published case where the periosteal proliferation and involucrum of osteomyelitis were mistaken for an ossifying subperiosteal hemorrhage of scorbutic origin. In the case reported by Benninghoven[3] the ulna of a ten month old infant was enveloped in a dense irregular periosteal proliferation such as is commonly seen in the osteomyelitis of infancy. I have not encountered an instance of scorbutic subperiosteal hemorrhage involving the ulna either in my immediate experience or in a perusal of the literature, and it seems very unlikely that such would occur inasmuch as the metaphyseal lesions of scurvy are minimal in the ulna and the ulnar epiphyses

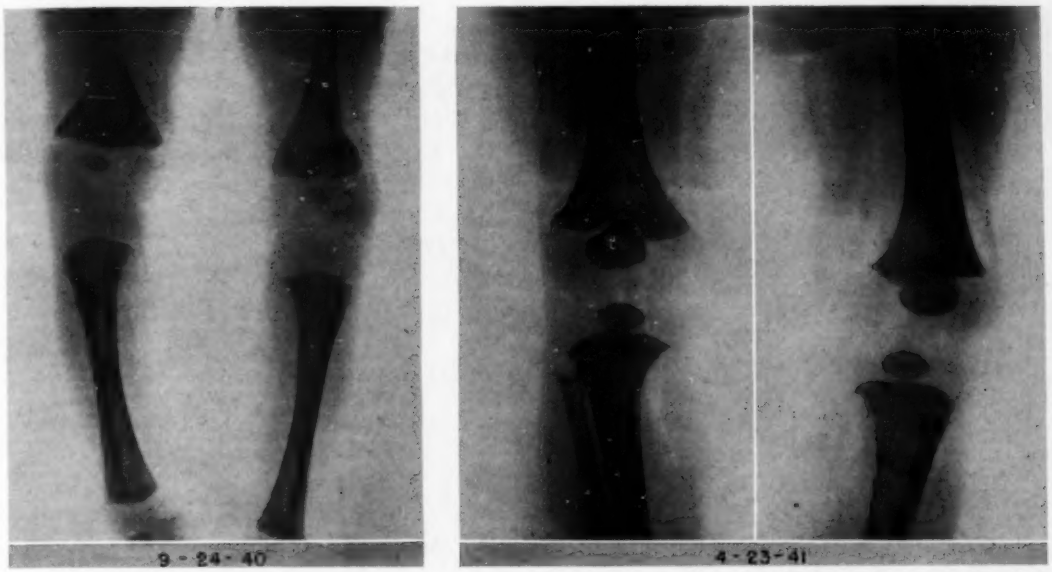

Fig. 7. Case v. H. R. A private patient of Dr. S. Bernstein, born about three weeks premature on August 22, 1940, of a difficult breech delivery. The legs were observed to be swollen and tender with mobility limited at the knees. The roentgen studies revealed a bilateral lateral dislocation of the distal femoral epiphyses and ossifying subperiosteal hemorrhages. Development and growth were very satisfactory, and the study at eight months shows no residual deformity.

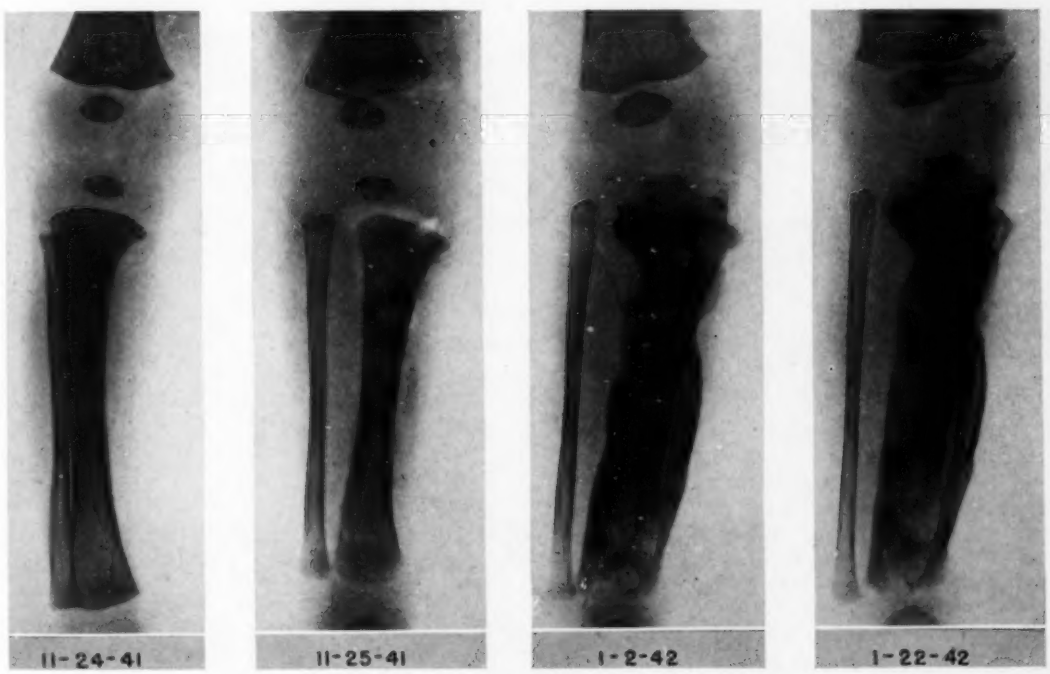

Fig. 8. Case vi. T. S. (CHM No. G7140). An eight week old infant admitted to the hospital with a story of multiple abscesses in the skin for two weeks. The tuberculin skin test and the serological tests for syphilis were negative. *Staphylococcus aureus* was obtained by blood culture. Treatment consisted in large doses of sulfadiazine and transfusions of blood. One of the sites of abscess formation was in the right knee, and the roentgenograms show the development of an osteomyelitis of the tibia with displacement of the proximal tibial epiphysis and extensive periosteal formation of new bone.

Fig. 9. Case vii. L. S. (CHM No. C7211). An eleven month old colored child with a long history of respiratory infection, diarrhea and feeding problems, and with an uncertain irregular intake of cod-liver oil and orange juice. There are the coarse trabecular markings, the laminated periosteal and cortical shadows in the shafts of the long bones characteristic of rickets. In addition there are submetaphyseal notchings (lower medial aspect of the left femur) and Type 1 periosteal shadows indicative of scurvy with early subperiosteal hemorrhage.

are undeveloped in infancy. My diagnosis of scurvy was based upon the finding of some blood by aspiration and a low vitamin C content of the blood. Both of these findings are quite compatible with osteomyelitis without scurvy. Benninghoven's opinion that subperiosteal calcification may precede typical scurvy changes in the diaphyses of the long bones is contrary to the experience of other observers.

In one case of this series (Case vi) there was a closer similarity to scurvy in that the ossifying periostitis was accompanied by epiphyseal displacement. The irregular density and contour of the periosteal shad-

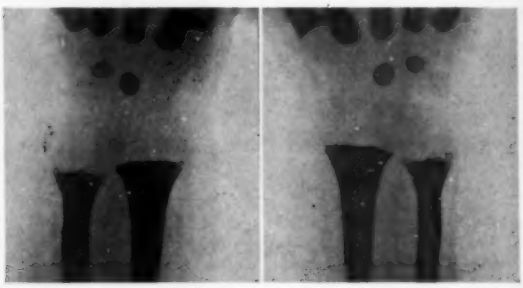

Fig. 10. Case viii. M. S. (CHM No. F3797). An eight month old white infant who had never been given orange juice or other antiscorbutic, and cod-liver oil only at irregular intervals. In the wrists the metaphyseal line at the lower end of the radius is dense and there is submetaphyseal notching characteristic of scurvy, while the flaring and cupping in the ulnar metaphyses indicate rickets.

ow is quite unlike that seen in scurvy, however. The destructive lesions in the bone itself as well as in bones elsewhere in the body, and the absence of any changes resembling scurvy in the metaphyses leave no doubt concerning the roentgen diagnosis of pyogenic osteomyelitis.

Trauma. Traumatic displacement of an epiphysis results in separation of the periosteum at the end of the shaft and subperiosteal hemorrhage not unlike that seen with the epiphyseal displacement of advanced scurvy and this lends some support to the view that the mechanism of the subperiosteal hemorrhage is the same in both instances. Epiphyseal displacements in infancy are usually the result of birth trauma and we have seen instances of subperiosteal hemorrhage apparently resulting from displacement of unossified proximal humeral

and femoral epiphyses. One instance, however, where the lower femoral epiphysis was displaced shows more clearly the relation of the epiphyseal displacement to the subperiosteal hemorrhage (Case v). It is true that the hemorrhage is not as profuse or extensive as that seen in scurvy, but there is, we think, an essential similarity in the manner by which the hemorrhage is produced. As in scurvy, there is amazingly little disturbance in the subsequent growth of the bone and no residual deformity or disability.

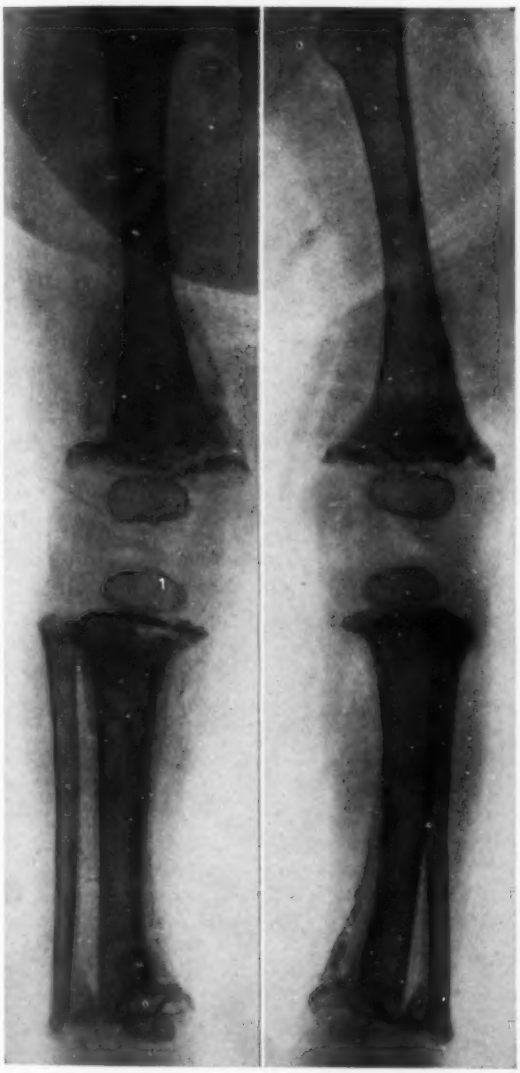

Fig. 11. Case ix. C. C. (CHM No. B7407). A nine month old white female nourished by formula feeding with no orange juice or cod-liver oil for four months and very small amounts before that. Advanced rachitic and scorbutic changes are seen in the metaphyses. There are laminated periosteal shadows of the rachitic type at the medial aspect of the tibial shafts, together with subperiosteal hemorrhages secondary to the scorbutic fragmentation, disintegration and displacement of the metaphyses.

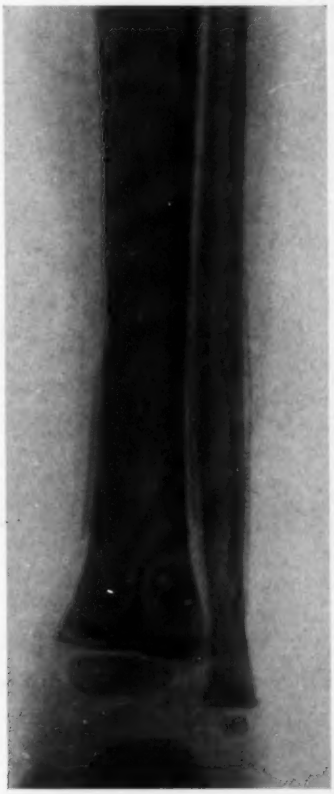

Fig. 12. Case x. F. D. (CHM No. F7952). Skeletal changes observed in a colored child, aged two, suffering from lymphatic leukemia with fever, malaise, and pains in the legs. The tuberculin skin tests were negative, as were the Kline and Kahn serological tests for syphilis. At the time of this study no abnormality could be detected in the blood other than a slight anemia. The leukemic picture appeared terminally seven weeks later and the diagnosis was confirmed at autopsy. The roentgen findings have certain resemblances to scurvy. The periosteal shadows occur away from the metaphysis, however, and there is infiltration of the underlying bone. The metaphyseal line is of increased width and density and there is submetaphyseal rarefaction, but without the notch representing minute fractures of the trabeculae seen in scurvy.

Neoplasm. Neoplastic infiltration, es-

pecially that of leukemia and neuroblastoma, may produce a picture similar to that of scurvy (Case x). A dense metaphyseal line with submetaphyseal rarefaction commonly occurs in leukemia as described by Baty and Vogt.[2] In scurvy the line is more sharp and dense, however, offering a greater contrast to the uniformly demineralized bone of the diaphysis. Also the submetaphyseal notch is recognizable as a distinct fracture rather than a band of demineralization such as occurs in leukemia. Subperiosteal leukemic deposits tend to occur about the mid-portions of the shafts of the long bones rather than in the metaphyseal areas and there is apt to be a recognizable infiltration or destruction in the underlying bone, as in Case x.

CONCLUSIONS

There is a close correlation between the clinical and roentgenological diagnosis of infantile scurvy. Pathognomonic signs are almost always demonstrable in roentgenograms made when the child is first seen in the clinic.

Infantile scurvy is still a common disease in hospital practice. It occurs almost exclusively in the narrow age range of seven to fifteen months.

The periosteal lesions of scurvy depend for their occurrence, location, and severity upon the severity of the lesions which develop in the metaphyseal areas of the long bones.

The differential diagnosis of scorbutic periosteal lesions from the periosteal lesions of rickets, infection, trauma and neoplasm is usually not difficult.

REFERENCES

1. Barlow, Thomas. On cases described as acute rickets. *Med. Chir. Trans.*, 1883, *67*, 159.
2. Baty, J. M., and Vogt, E. C. Bone changes of leukemia in children. Am. J. Roentgenol. & Rad. Therapy, 1935, *34*, 310–314.
3. Benninghoven, C. D. Subperiosteal calcification and bone-changes in early scurvy. *California & West. Med.*, 1939, *50*, 278–279.
4. Bromer, R. S. Roentgen-ray diagnosis of infantile scurvy. Am. J. Roentgenol. & Rad. Therapy, 1928, *19*, 112–125.
5. Chiari, O. Ueber periostale Reaktionen bei nicht luetischen Kindern im ersten Trimenon. *Acta paediat.*, 1937, *22*, 436–440.
6. von Chiari, O. Ueber periostale Knochenveränderungen im frühen Saüglingsalter. *Arch. f. Kinderh.*, 1938, *115*, 149–168.
7. Follis, R. H., Jackson, D. A., and Park, E. A. Problem of association of rickets and scurvy. *Am. J. Dis. Child.*, 1940, *60*, 745.
8. Fraenkel, E. Untersuchungen über die Moeller-Barlowsche Krankheit. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1903–1904, *7*, 231; 291.
9. Lind, James. A Treatise on the Scurvy. Second edition. 1757, p. 260.
10. McLean, S., and McIntosh, R. Healing in infantile scurvy as shown by x-ray. *Am. J. Dis. Child.*, 1928, *36*, 875–930.
11. Meier, A. Periostitis hyperplastica bei einem 3 Monate alten Frühgeborenen. *Arch. f. Kinderh.*, 1936, *109*, 223–228.
12. Nelson, W. E., Doughty, W. M., and Mitchell, A. G. Roentgenographic visualization of subperiosteal hemorrhage in infantile scurvy. *J. Am. M. Ass.*, 1933, *101*, 14–17.
13. Park, E. A., Guild, H. G., Jackson, D., and Bond, M. Recognition of scurvy with especial reference to early x-ray changes. *Arch. Dis. Childhood*, 1935, *10*, 265–294.
14. Pelkan, K. F. Roentgenogram in early scurvy. *Am. J. Dis. Child.*, 1925, *30*, 174–188.
15. Wimberger, H. Zur Diagnose des Saüglingsskorbuts. *Ztschr. f. Kinderh.*, 1923, *36*, 279–285.

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ROENTGENOGRAPHIC DEMONSTRATION BY TANTALUM POWDER OF SINUSES RESULTING FROM EXTRACTION OF INTERVERTEBRAL DISC PROTRUSIONS*

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IN 1939 we began implanting a silver hemostatic clip as deeply as possible into the sinuses resulting from the protrusion of intervertebral discs, as Barr[1] had suggested two years earlier. This served to identify the level of the lesion accurately by roentgenography when the operation was done without the removal of bone and to confirm possible recurrence by displacement of the clip (Fig. 1). The blind closure of the clip in the sinus cannot insure that it is attached to residual disc material, so its subsequent displacement is not specific in meaning.

FIG. 1. Displacement of silver clip from disc sinus by recurrent disc protrusion. *A*, clip placed in sinus after removing protruded portions. *B*, displacement of clip upon recurrence of symptoms. Recurrence of protrusion confirmed surgically.

Attempting to visualize the postoperative alterations in the disc sinus following curettage of all easily removable fragments, in several cases the sinus was filled with lipiodol or with a colloidal suspension of thorium dioxide (thorotrast), the excess of which was removed by suction. Not enough of the material adhered to the sinus walls for satisfactory roentgenograms. Sterile tantalum powder† then was tried for this purpose. We know of no previous use of tantalum powder as a contrast medium in the body, but we have used it also to demonstrate the extent of the incisions in prefrontal lobotomy. One curette cup (6 by 4

† Supplied by Mr. I. R. McCall, Fansteel Metallurgical Corporation, Chicago, Illinois.

* From the Buffalo General Hospital and University of Buffalo School of Medicine.

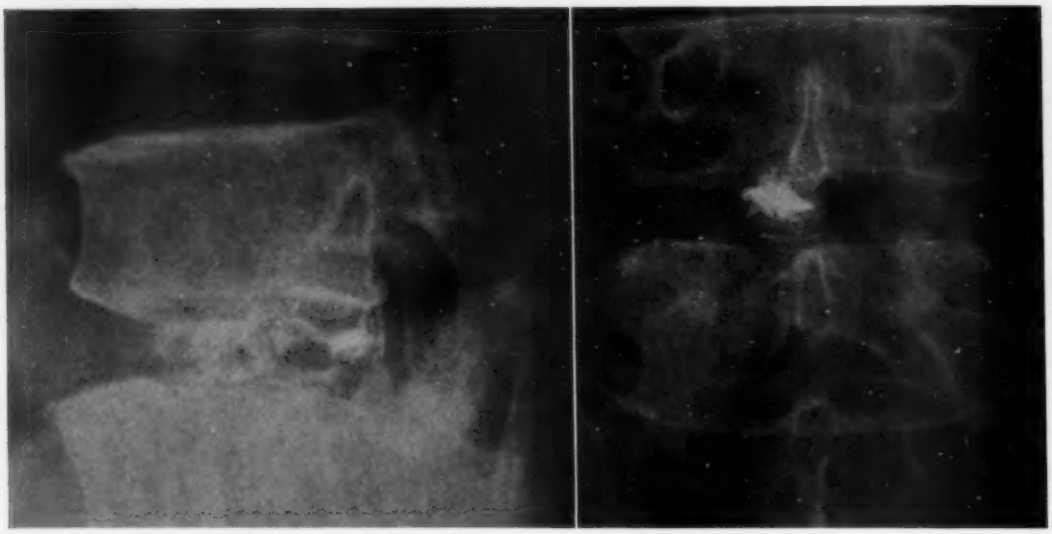

FIG. 2. Disc sinus demonstrated by tantalum powder, ten days after operation.

by 2 mm.) full of the powder is introduced into the disc sinus, being careful to allow none to come into contact with the tissues of the spinal canal. With the curette, it is dispersed as widely as possible within the disc. At present, double this amount of powder is used, since the small amount does not produce a sufficiently dense lateral roentgenogram. Stereoscopic roentgenograms are made of the operated area prior to discharge from the hospital. Since September, 1943, this procedure has been employed in approximately 25 selected cases. In a few, additional roentgenograms were made several months later to see if activity had altered the appearance of the sinus.

OBSERVATIONS

The demonstrated disc sinus resembles an elongated slit with an irregular cross

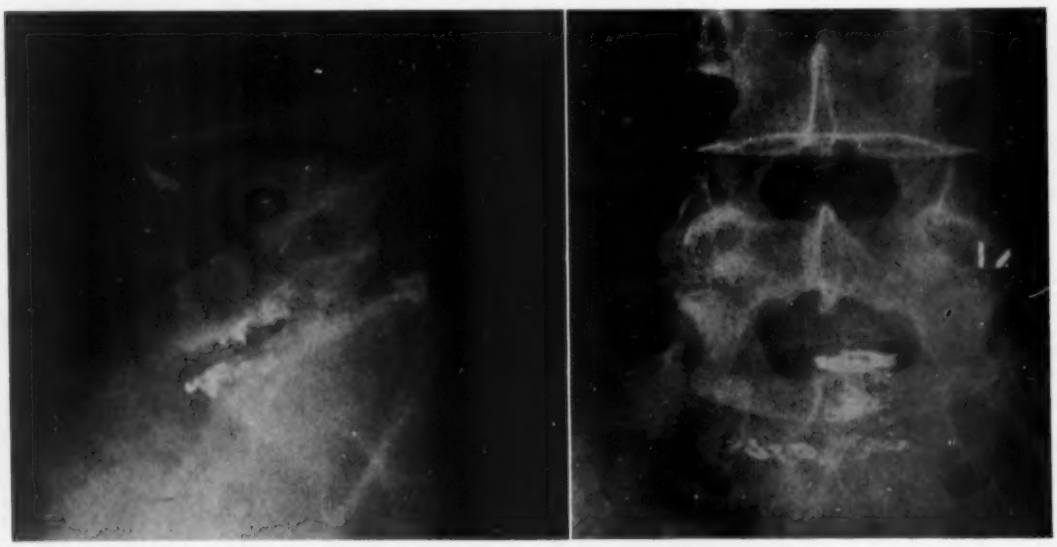

FIG. 3. Demonstration of tantalum very near the anterior annulus. Note lateral extent of the disc defect in the anteroposterior view. (The silver clips resulted from an earlier sympathectomy.)

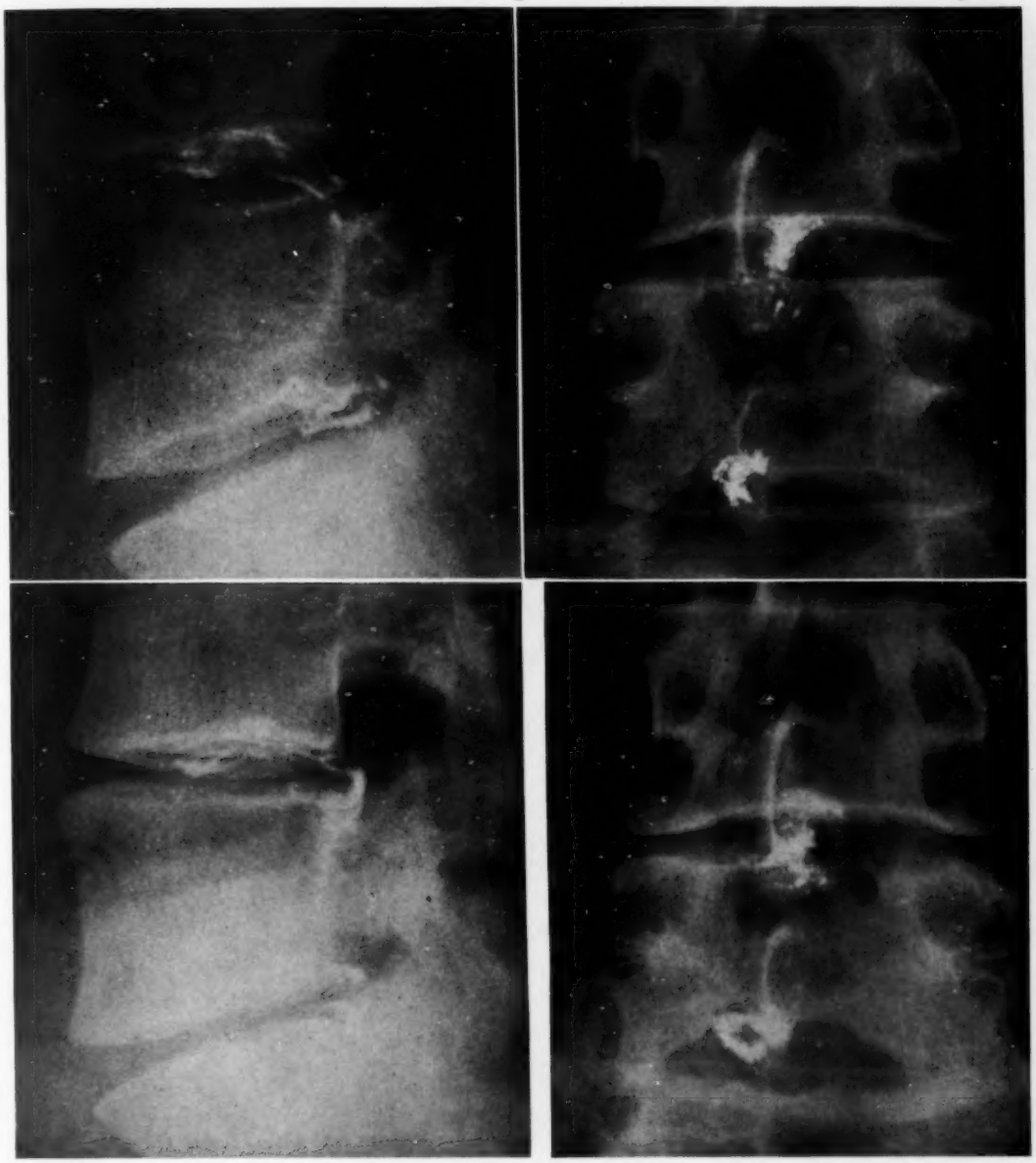

Fig. 4. *Above*—Views made after removal of disc protrusions from fourth and fifth lumbar discs. *Below*—Views made seven months later. Only minimal changes are apparent.

section (Fig. 2). Occasionally the shadow will suggest a round or oval cavity. In one case (Fig. 3) the opaque material extended to the anterior edge of the vertebral bodies, suggesting that the curette had approached the annulus fibrosus. This warns that curettage of the disc must be done cautiously to avoid penetration of the annulus. This has occurred in a few cases, with damage to retroperitoneal structures, as mentioned by Love.[2]

Roentgenograms made after seven months of normal activity following the original (Fig. 4) show no significant alterations. We have had no opportunity to re-examine after tantalum implantation, a case with symptoms suggesting a recurrence of protrusion.

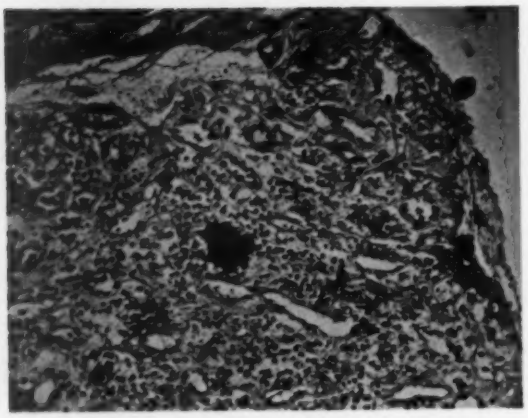

FIG. 5. Tantalum powder within a granulation removed six weeks after implantation of the powder. There is no increase in reaction in the immediate neighborhood of the powder.

SEQUELAE

Such implantation of tantalum powder has resulted in no untoward sequelae. The operative area has been re-examined in one case. A typical disc protrusion was exposed by interlaminal approach through the fifth lumbar interspace and 3.5 cc. of disc material was removed with the curette. Tantalum powder was introduced as described above. Six weeks later the patient displayed signs of a contralateral lesion and since she had other evidence of an unstable lumbosacral joint, re-exploration and fusion was advised. A protrusion of the same disc was removed on the opposite side. The site of the original operation then was exposed. The sinus into the disc was patent. Curettage obtained 1.5 cc. of yellowish-pink material through which could be seen conglomerate and scattered fragments of opaque black material. Histopathologic study showed (Fig. 5) mild degenerative and regressive changes with some compact collagen and granulation tissue associated with a few bone fragments. Within some of the granulations were several small particles of black foreign material, near some of which were a few multinucleated cells suggesting foreign body giant cells. The tissue reaction was not increased around the tantalum fragments. In other areas where the powder was in contact with simple disc material, rather than in granulation tissue, practically no tissue reaction was evident.

SUMMARY

(1) Following removal of protrusions of lumbar intervertebral discs, the resulting sinuses have been visualized by roentgenography, with tantalum powder as an opaque medium.

(2) The sinus appears as a rather narrow defect extending toward, and in some cases to, the anterior part of the annulus fibrosus.

(3) No significant alteration in the appearance has developed after seven months of normal activity of the patient.

(4) No untoward sequelae have followed mplantation of the tantalum powder.

(5) Histopathologic study, in one case, of tissue removed six weeks after implantation of the powder, showed only mild reactive changes.

REFERENCES

1. BARR, J. S. "Sciatica" caused by intervertebral-disc lesions; report of 40 cases of rupture of intervertebral disc occurring in low lumbar spine and causing pressure on cauda equina. *J. Bone & Joint Surg.*, 1937, *19*, 323–342.
2. LOVE, J. G. Differential diagnosis of intraspinal tumors and protruded intervertebral disks and their surgical treatment. *J. Neurosurg.*, 1944, *1*, 275–290.

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ANOMALOUS DEVELOPMENT OF THE FIRST RIB SIMULATING ISOLATED FRACTURE

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ISOLATED fractures of the first rib are infrequently reported. The earliest description of a fracture of the first rib was presented by Jones[6] in 1869. In the beginning of the twentieth century 10 more cases were reported—all discovered at autopsy. Lane[8] found 4 cases in 200 autopsies, and reported them in 1887. More recently, isolated fractures of the first rib have been discovered by use of the roentgenogram. In 1934, Huber[4] reported 22 cases of isolated fractures of the first rib in an extensive search of the literature and added 4 of his own. Two years later, Breslin,[2] apparently unaware of Huber's paper, also compiled 22 cases from the literature, but some of these cases were not the same as those in Huber's group. Breslin added 5 cases of his own. In recent years, probably the most exhaustive investigation of the literature on this subject was undertaken by Sjögren[9] in 1942. After a careful perusal of the papers of Huber and Breslin, and a painstaking analysis of all available recent papers on the subject, he decided that the total number of cases of isolated first rib fractures reported in the medical literature was between 60 and 70, including 5 cases of his own. This clearly indicates the relative infrequency of these fractures.

ANATOMY AND MECHANISM OF OCCURRENCE

Before discussing the possible mechanism of isolated fractures of the first rib a brief description of the anatomy of the first rib is in order.

The first rib is deeply situated in the neck, approximately in its center, and is very adequately protected by the clavicle anteriorly and the scapula and the muscles of the shoulder girdle posteriorly. It is the shortest of all the ribs, and is flat and broad, with a superior and an inferior surface and an outer border. The superior surface has two shallow depressions separated by the tuberculum scaleni to which is attached the scalenus anticus muscle. The subclavian vein passes over the groove in front of the tuberculum scaleni, and the subclavian artery passes behind it. The scalenus medius muscle attaches to an area between the groove for the artery and the tuberculum scaleni. Part of the serratus magnus muscle attaches to the posterior portion of the outer border of the first rib. Thus, three muscles attach to this rib. The first rib articulates anteriorly through its costal cartilage with the sternum and posteriorly with the first thoracic vertebra.

From the above description, and after due consideration of the protective factors involved, it would seem extremely unlikely that direct trauma could affect the first rib alone. What, then, is the mechanism of isolated fracture of the first rib? Sjögren enumerates three possibilities: (1) indirect trauma—the force being conducted through the clavicle; (2) direct trauma—the force being conducted through the back, and (3) indirect trauma—the force being transmitted through the manubrium sterni. The first-named mechanism he considers by far the most common. In this, the shoulder suddenly moves downward and backward and the clavicle strikes the first rib which is fixed by the scalenus anticus muscle. He likens the clavicle to a fulcrum. But Sjögren admits that his explanation (also promulgated by Lane[8] and Johnston[5] fails to explain many published and observed

cases of isolated fractures of the first rib, particularly where there is no history of trauma to the upper chest. In recent years the scalenus anticus muscle has been implicated in spontaneous isolated fractures of the first rib where trauma has played no rôle.[7] It is supposed that sudden stress with the scalenus anticus muscle contracting may cause such fractures.

FIG. 1. Callus-like change in left first rib.

FIG. 2. Callus-like change in left first rib.

We became interested in this topic in August, 1943, when we began to observe changes simulating fractures in the first rib on the chest roentgenogram. From August, 1943, until February, 1944, a period of six months, we have, in our routine work, discovered 17 cases in which the first rib has shown changes which could be interpreted as being due to fracture. During this period we have made 62,782 roentgenographic chest examinations. This means an incidence of 1 in 3,692. Of our 17 cases, 14 have been in Negroes and 3 in the white race. All have been males, with the age group ranging from eighteen to thirty-two.* In 7 cases the right first rib was involved, and in 10 cases the change was noted in the left first rib. We have observed two distinct types of change in our 17 cases. In 7 cases there was an appearance simulating callus formation about a fracture site. In 9 cases there was a change which we have chosen to call pseudarthrosis. (It should here be noted that fractures of the first rib are said to heal either by callus formation or by the appearance of pseudarthrosis as the end stage,

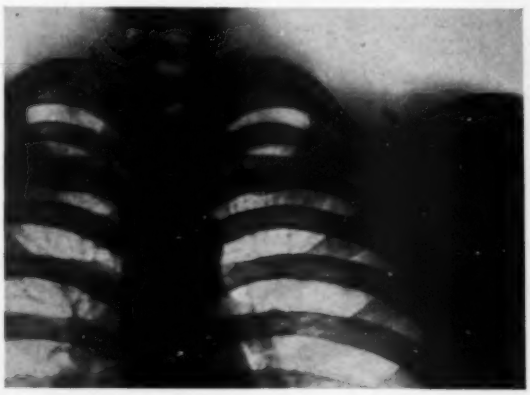

FIG. 3. Callus-like change in left first rib.

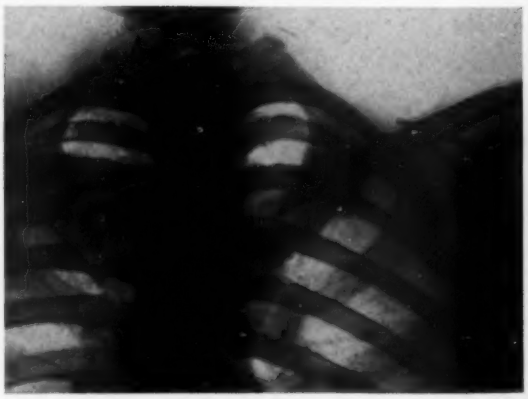

FIG. 4. Callus-like change in left first rib.

* It should be kept in mind that we examine many more Negroes than white persons because of our Induction Station work. Also, we examine relatively few women on this Post in comparison with the number of men examined.

with the former predominating.[8,9]) In 1 case we have felt that the changes were a combination of callus-like appearance and pseudarthrosis. In Figures 1–4 we are presenting 4 examples of callus-like change. Figures 5–8 are illustrations of pseudarthrosis-like appearance. Figure 9 is our 1 example of combined change, i.e. callus and pseudarthrosis.

All of our cases were closely questioned as to past history with particular reference to the possibility of trauma or sudden onset of pain in the area of the first rib. In 16 of the 17 cases there was absolutely no history of injury, even remotely connected with the area involved, nor was there anything to suggest a sudden onset of pain such as might occur with spontaneous fractures. In only 1 case was there a history of trauma—an automobile accident six years previously, with a diagnosis by the attending physician of rib fractures of the right upper chest (the side involved by

Fig. 5. Pseudarthrosis-like change in left first rib.

Fig. 6. Pseudarthrosis-like change in right first rib.

Fig. 7. Pseudarthrosis-like change in left first rib.

Fig. 8. Pseudarthrosis-like change in right first rib.

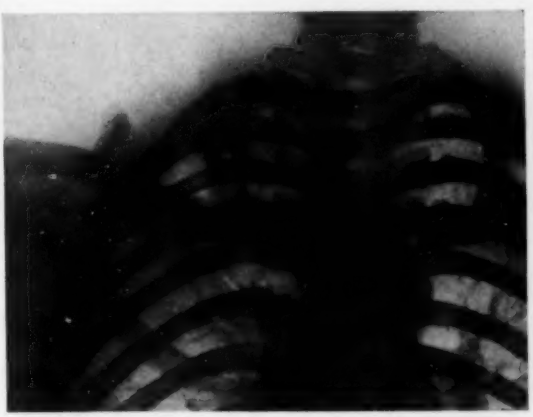

Fig. 9. Combined changes in right first rib.

change in the first rib). The change in this case appeared to be one of pseudarthrosis with no evidence of fracture to the other ribs. We have, to date, been unable to consult with the attending physician, or to obtain previous chest roentgenograms on this individual.

In none of our cases was there any evidence of fracture, old or recent, involving the other ribs.

DISCUSSION

We do not feel that any of these cases have had fractures of the first rib, either spontaneously by scalenus anticus muscle pull, or by the direct and indirect methods due to trauma, enumerated above. In the 1 case with a history of injury the pseudarthrosis-like change does not differ materially from the 8 other cases showing similar changes. Since none of the other ribs in this individual show evidence of old fracture, our opinion is that no fracture of the first rib was ever present in this individual. It is quite possible that after the automobile accident roentgenograms showed the defect in the first rib and fracture was diagnosed on this basis.

We are of the definite conviction that in all of our cases the changes, as demonstrated, are due to anomalous development of the first rib. The possibility of such development is not stressed in the American literature, and in the literature available to us it is not even mentioned. The foreign literature that we have been able to obtain is quite vague on the subject, and where anomalous development is mentioned, these anomalies occur at the costochondral junction and cannot be mistaken for fracture. In this connection it is interesting to note that the skeleton of the bird contains a joint on the first rib. We would like to point out that such anomalies may occur in the clavicle as shown recently by de Lorimier.[3] He calls them "mesenchymal articulations." From the roentgenographic reproduction in de Lorimier's book it is evident that the defect in each clavicle in his case closely simulates our group of cases with pseudarthrosis-like appearance.

We are not suggesting that isolated fractures of the first rib, as heretofore reported in the literature, do not occur. On the contrary, many of the case reports that we have reviewed seem to authentically represent such fractures, with a definite history of injury to the area involved. Even here we are not convinced that all cases suffering trauma with a resultant diagnosis of first rib fracture actually represent fractures. It is readily apparent that such deformities as we have described might be called fractures, particularly in the wake of trauma. Most observers writing on the subject of isolated first rib fractures admit that, considering the anatomical make-up and protective features of the first rib, such fractures should occur only infrequently, since trauma severe enough to fracture the first rib should also fracture other parts of the thoracic cage and its surrounding bony structures. As for spontaneous isolated first rib fractures with no history of trauma, we believe that the reported cases should lend themselves to closer surveillance with the probability existing that a number represent anomalous first rib development. The explanation usually given for such spontaneous fractures is sudden contraction of the scalenus anticus muscle during great stress. We find it difficult to believe that a muscle pull could easily fracture a short, broad and relatively strong bone such as the first rib. It is even said that such spontaneous fractures may occur without symptons. Thus, Axen in a personal communication to Sjögren[9] stated that he found spontaneous isolated fractures of the first rib in 4 out of 17,000 individuals presenting themselves for routine chest roentgenograms—all of them asymptomatic. Interestingly enough, this is an incidence of 1 in 4,222 cases, which is comparable to our incidence of 1 in 3,692. It is difficult for us, at best, to accept the "scalenus anticus muscle pull" theory of spontaneous fracture. It is impossible for us to accept the notion that it may be asymptomatic when it does occur in an otherwise healthy individual. We

have not seen the roentgenograms on Axen's cases but we make bold to state that they could very well represent the anomalous development that we believe we are dealing with.

The incidence of occurrence of this anomaly (1 in 3,692 cases in our series) indicates that we are dealing with a finding that will be seen relatively frequently, particularly by those dealing with large groups of cases, i.e. mass public health surveys, Armed Forces Induction Station examinations, etc. It is important that such cases be correctly identified as anomalies of the first rib and not fractures.

We are of the opinion that this subject is of some interest, both from an academic point of view and from the medicolegal aspect. Undoubtedly, litigation has or will come up in relation to isolated first rib fractures and the probability that the roentgenographic changes may be developmental instead of representing fracture should always be borne in mind.

SUMMARY AND CONCLUSIONS

1. A brief survey of the literature regarding isolated fractures of the first rib is presented.

2. The anatomy of the first rib is discussed and the possible mechanisms for first rib fractures are described.

3. Reproductions of roentgenograms of 9 out of a series of 17 personally observed cases of first rib changes simulating fracture are included.

4. The hypothesis that these changes (callus-like formation and pseudarthrosis) are due to anomalous development is amplified.

5. The frequency of occurrence of these changes is emphasized and the probability that these apparent congenital changes may be mistakingly diagnosed as isolated first rib fractures—particularly of the "spontaneous" variety—is introduced.

REFERENCES

1. Aitken, A. P., and Lincoln, R. E. Fracture of first rib due to muscle pull. *New England J. Med.*, 1939, *220*, 1063–1064.
2. Breslin, F. J. Fractures of first rib unassociated with fractures of other ribs. *Am. J. Surg.*, 1937, *38*, 384–389.
3. de Lorimier, A. A. The Arthropathies. Year Book Publishers, Inc., Chicago, 1943, p. 39.
4. Huber, P. Ueber die Fraktur der 1. Rippe. *Arch. f. klin. Chir.*, 1934, *179*, 280–289.
5. Johnston, G. M. *Dublin. J. M. Sc.*, 1896, *102*, 191. Quoted by Breslin.[2]
6. Jones, T. E. *Brit. M. J.*, 1869, *2*, 201. Quoted by Breslin.[2]
7. Kohlbach, W. Verschiedenartige Kontinuitätstrennungen der 1. Rippe. *Röntgenpraxis*, 1939, *11*, 626–629.
8. Lane, W. A. *Brit. M. J.*, 1887, *2*, 119. Quoted by Breslin.[2]
9. Sjögren, S. E. Isolierte Frakturen der ersten Rippe. *Acta radiol.*, 1942, *23*, 79–89.
10. von Sassen, W. Pseudarthrose nach isolierter Fraktur der 1. Rippe. *Deutsche Ztschr. f. Chir.*, 1938, *250*, 76–79.

THE TRITICEOUS CARTILAGES*

A ROENTGEN-ANATOMIC STUDY

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Medical Corps, Army of the United States

THE correlation between the shadows seen on roentgenograms of the neck and the normal anatomy has been well established. This is especially true in the case of the laryngeal cartilages, which when ossified lend themselves particularly well to roentgen visualization because of the contrast produced between them and the adjacent soft tissues and air-filled spaces.

This paper deals with the triticeous cartilages, small, round or oval bodies of cartilage in the lateral hyothyroid ligament. These cartilages may undergo ossification and have been seen and identified on roentgenograms of the neck (Taylor,[5] Picchio,[2] Shanks, Kerley and Twining[4] and others). It is the purpose of this paper to illustrate unusual types of development which may occur in these cartilages.

The location and relationships of the triticeous cartilages is shown in Figure 1*C*. The mode of development of these carti-

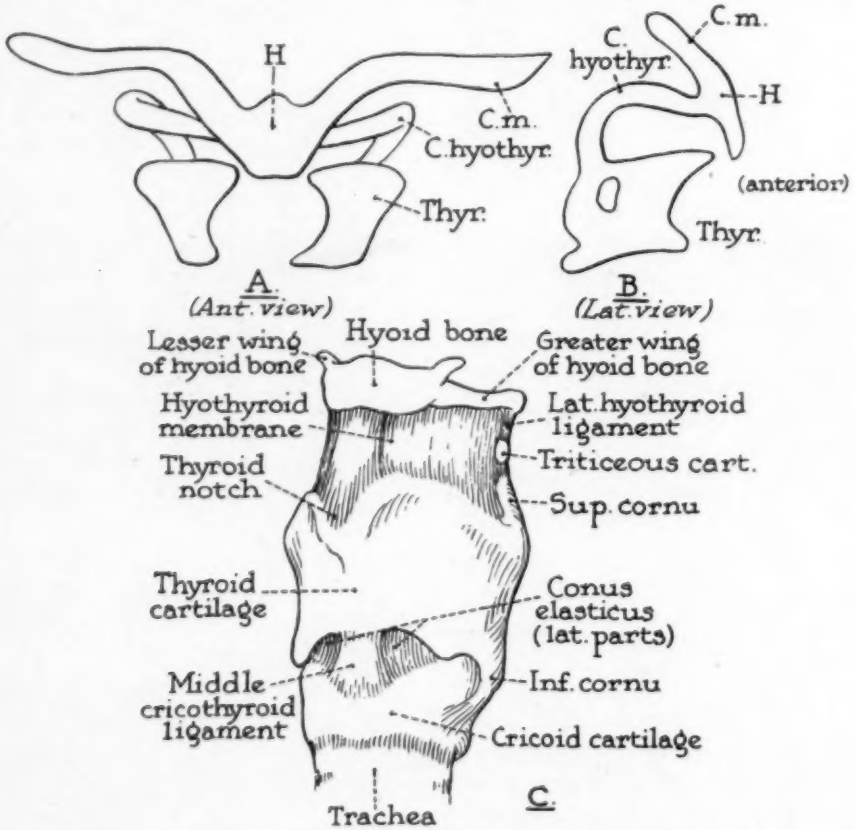


FIG. 1. *A* and *B*, the state of development of the thyroid cartilage and hyoid bone in a forty day old embryo (after Kallius from Kiebel and Mall[1]). *H*, body of hyoid bone; *C. m.*, lesser wing of hyoid bone; *Thyr.*, thyroid cartilage; *C. hyothyr.*, hyothyroid cartilage.

C, relation of the triticeous cartilage to the laryngeal cartilages and associated membranes and ligaments, and the hyoid bone. Oblique view.

* All the material for this paper was obtained while the author was a resident at the University of Illinois College of Medicine, Chicago, Illinois. From the Departments of Radiology and Physiology.

lages may be briefly summarized as follows:

Early in fetal development, the body of the hyoid bone and the lamina of the thyroid cartilage are joined by a curved cartilaginous bar, the hyothyroid cartilage. Ventrally this bar is joined to the posterior aspect of the body of the hyoid bone, and below and dorsally it becomes fused to the cartilage of the lamina of the thyroid cartilage (Fig. 1, *A* and *B*). The hyothyroid cartilage represents the skeleton of the third and fourth visceral arches. With further development the parts become molded so that the ventral portion becomes the greater wing of the hyoid bone, and the inferior dorsal portion becomes the superior horn of the thyroid cartilage. These parts remain connected by cartilage until the third month when they become discontinuous, being separated by a small island of cartilage (the triticeous cartilage) lying in connective tissue (the hyothyroid ligament). This ligament does not reach its full stage of development until after birth, when the hyoid bone and thyroid cartilage become further separated.

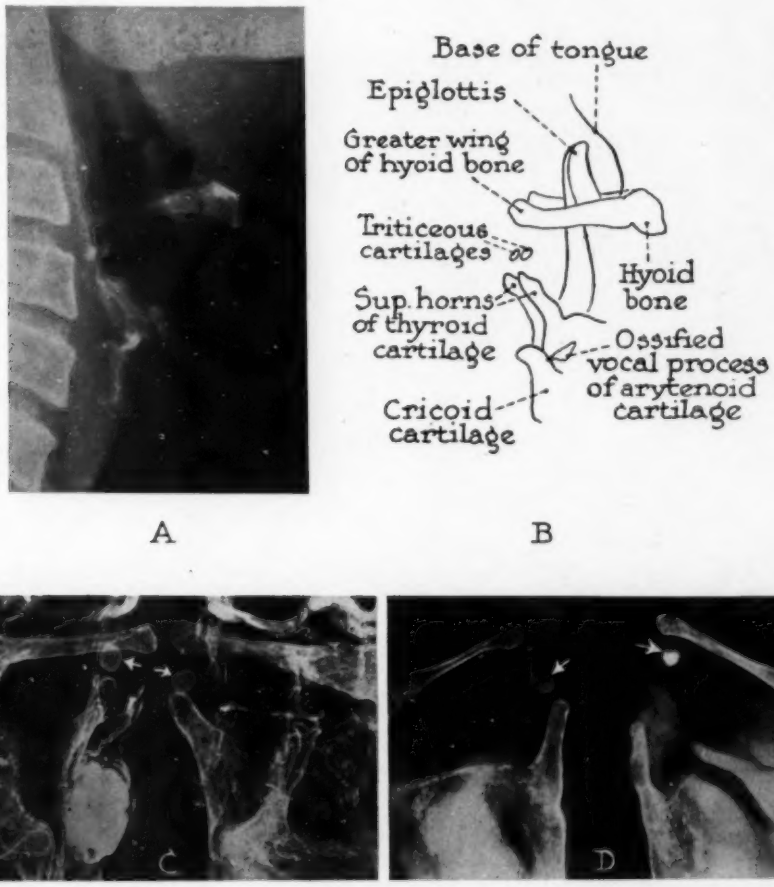
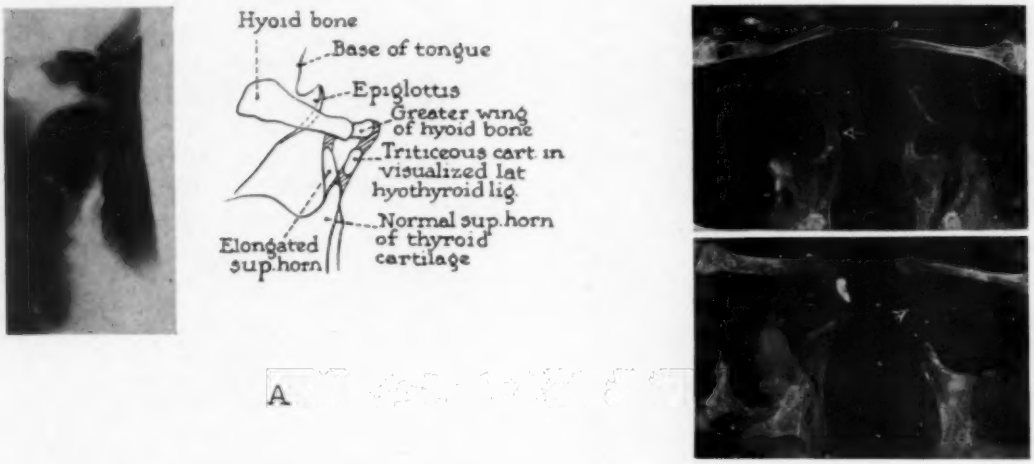


Fig. 2. *A* and *B*, roentgenogram and diagram illustrating a case of bilaterally developed symmetrical triticeous cartilages, showing a similar type of ossification.

C and *D*, roentgenogram of cadaver specimens to show bilaterally developed triticeous cartilages. In *C* the cartilages show a similar type of ossification. In *D* the ossification is dissimilar.

The usual mode of development results in bilateral and symmetrical cartilages, one in each hyothyroid ligament. When ossified, the roentgen appearance of these is as shown in Figure 2, *A*, *B* and *C*. Occasionally the ossification may vary on the two

sides (Fig. 2 *D*). Bilaterally developed cartilages which do not ossify are not seen on the roentgenogram. A similar roentgen appearance is obtained in those instances in which the cartilages do not develop.



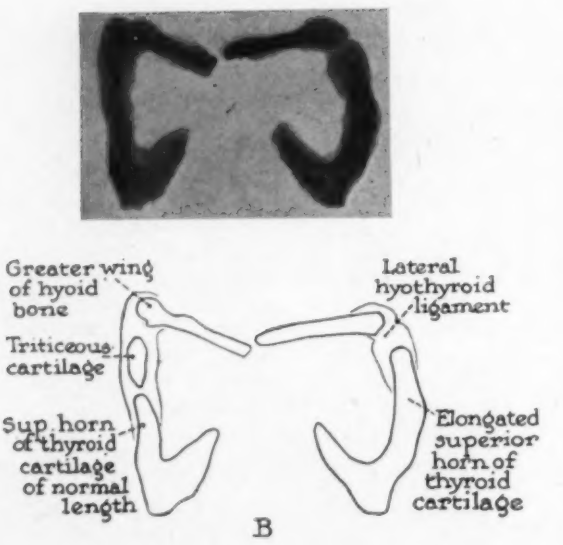

Greater wing of hyoid bone
Triticeous cartilage
Sup. horn of thyroid cartilage of normal length
Lateral hyothyroid ligament
Elongated superior horn of thyroid cartilage
B

FIG. 3. *A*, lateral roentgenogram of the neck and diagram showing a unilateral ossified triticeous cartilage. The superior horn of the thyroid cartilage on the side of the absent triticeous cartilage is elongated, its length being equal to the combined length of the superior horn of the thyroid cartilage and triticeous cartilage on the other side. The roentgenogram was made during performance of the Valsalva procedure. The soft tissue shadow of the hyothyroid ligament was clearly seen and is shown by the shaded areas on the diagram accompanying the reproduction of the film.

B, roentgenogram and diagram of postmortem specimen of case shown in *A*.

C and *D*, roentgenograms of cadaver specimens showing instances of unilateral development of the triticeous cartilage. In *C* the probable point of fusion or lack of separation of the triticeous cartilage and superior horn of the thyroid cartilage is indicated by an arrow. In *D* the superior horn of the thyroid cartilage on the side of the absent triticeous cartilage is unossified superiorly. Its tip is indicated by an arrow.

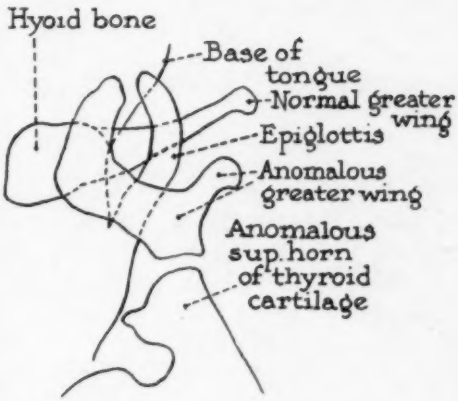
The triticeous cartilage may be present on one side only and be either cartilaginous or ossified. In cartilages which are ossified, the roentgen appearance is interesting. The superior horn of the thyroid cartilage on the side of the absent triticeous cartilage is elongated compared to the opposite superior horn. The length of the elongated horn is approximately equal to the combined length of the opposite superior horn plus the developed triticeous cartilage (Fig. 3). The probable explanation of this appearance is that in development one triticeous cartilage does not separate inferiorly but remains attached to the cartilage destined to form the superior horn of the thyroid cartilage on that side.

During the process of formation of the triticeous cartilage, irregularities in seg-

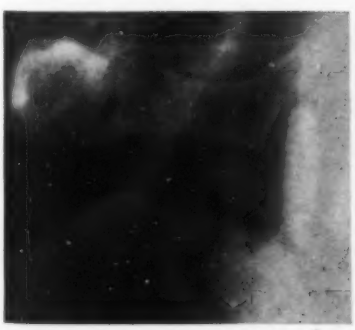
Fig. 4. Roentgenogram of a cadaver specimen showing a small accessory ossified triticeous cartilage (arrow).

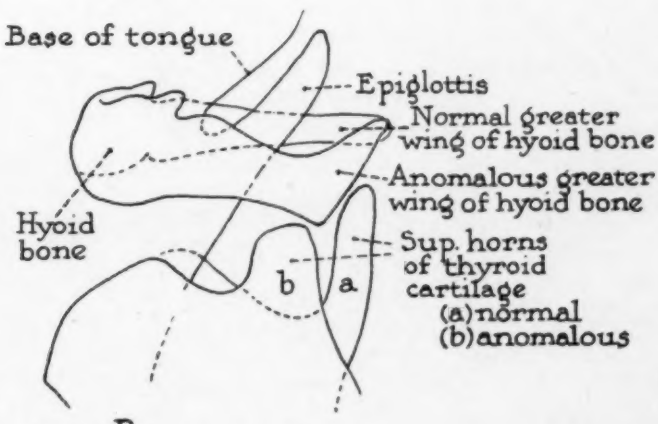
mentation of the fetal hyothyroid cartilage probably explain accessory cartilages in the adult hyothyroid ligament, such as illustrated in Figure 4.

An unusual type of structure in the region being discussed is that shown in Figure 5, which probably represents an arrested stage of development. Three such cases were found in adults. Instead of the usual elongated slender appearance, the superior horn of the thyroid cartilage is blunted and widened. The greater wing of the hyoid bone is broadened and posteriorly dips down toward the altered superior horn



A





B

Fig. 5, *A* and *B*. Roentgenograms and drawings to show two instances of a type of anomaly which probably represents that stage in development in which the hyoid bone and thyroid cartilage are joined by the hyothyroid cartilage. In *A* the normal superior horn of the thyroid cartilage is not ossified. In *B* it is ossified.

of the thyroid cartilage. The two are separated by a short gap which may be occupied by either cartilage or fibrous tissue. All 3 cases were found in roentgenograms of patients. No postmortem cases of this anomaly were found and therefore the type of tissue in the unossified gap could not be determined. This anomaly resembles that stage in the development of the hyoid bone and thyroid cartilage in which they are joined by the hyothyroid cartilage. In all 3 instances the anomaly was unilateral.

FIG. 6. *A*, drawing of Puchowski's case showing bilaterally ossified hyothyroid ligaments and an anomalous ossified styloid process extending from the base of the skull to the hyoid bone. *B*, roentgenogram and diagram of cadaver specimen showing an ossified superior horn of the thyroid cartilage similar to those of Figure 5. The greater wing of the hyoid bone, however, is normally developed. The hyothyroid ligament is almost entirely ossified.

Puchowski[3] described a case of ossified hyothyroid ligaments (Fig. 6*A*). A roentgenogram of a similar case is shown in Figure 6*B*. Only half of the specimen was available. Puchowski's case was bilateral and associated with complete ossification of the styloid process, which is not unusual, and which also is an anomaly of development representing a stage in the normal evolution of the styloid process. The superior horn of the thyroid cartilage of the specimen shown in Figure 6*B* is similar to those described above and illustrated in Figure 5. However, unlike the previous anomaly described, the hyoid bone is normal, and the hyothyroid ligament is almost completely ossified.

SUMMARY

The roentgen anatomy, both of the normal, and of the anomalies of development, of the triticeous cartilages and adjacent structures, has been discussed. The anomalies have been correlated with the normal development of the parts. This information is of value in the correct interpretation of roentgenograms of the neck, especially in explaining unusual findings in the region of the triticeous cartilages.

I am thankful for the suggestions and guidance of the late Dr. A. Hartung and Dr. G. E. Wakerlin, Heads of the Departments of Radiology and Physiology, respectively, and of Dr. T. J. Wachowski, Assistant Professor of Radiology, of the University of Illinois, College of Medicine, Chicago, Illinois.

REFERENCES

1. KIEBEL and MALL. Manual of Human Embryology. J. B. Lippincott Co., Philadelphia, 1910.
2. PICCHIO, C. Appunti critici di anatomia radiografica e di semeiologia radiologica del collo. *Radiol. med.*, 1935, *22*, 881–914.
3. PUCHOWSKI, B. Un cas d'anomalie de l'appareil hyoïdien. *Acta oto-laryng.*, 1933, *18*, 447–452.
4. SHANKS, S. C., KERLEY, P., and TWINING, E. W., Editors. A Text-Book of X-Ray Diagnosis. By British Authors. Vol. 1, p. 172. H. K. Lewis & Co., Ltd., London, 1938.
5. TAYLOR, H. K., and NATHANSON, L. Roentgen demonstration of cysts in the upper air passages. *Ann. Otol., Rhin. & Laryng.*, 1935, *44*, 170–180.

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CHANGES AFTER TREATMENT OF AN UNPROTECTED BRAIN WITH LARGE DOSES OF ROENTGEN RADIATION*†

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THE effect of roentgen irradiation on the brain is the same whether one irradiates the skull-protected or the unprotected brain. These are at least the conclusions which may be drawn from the investigations of Lyman, Kupalov and Scholz,[5] of Scholz,[12] and of Davidoff Dyke, Elsberg and Tarlov[1] who treated dogs and monkeys with large doses of roentgen rays.

As stressed particularly by Marburg and Sgalitzer,[7] it seems that most of the roentgen changes in the brain are caused by vascular lesions. As for the kind of these lesions in the arteries as well as in the veins, Windholz,[14] among others, claimed changes in the blood vessel walls themselves, whereas Mogilnitzky and Podljaschuk[8,9] stressed especially the changed permeability of the vessel walls. Thus there is apparently, in addition to the anatomical lesion, a functional one as also indicated by Scholz.[12] This helps to explain the relatively late occurrence of the signs in the irradiated animals.

Experiences concerning the effect of irradiation on the unprotected human brain with large doses of roentgen rays are lacking in spite of the treatment of 18 such cases by Elsberg, Davidoff and Dyke.[3,4] Five of the patients died within twelve hours to eleven days after treatment, a time relatively too short to allow any judgment of the roentgen irradiation effect.

Therefore it seems to be justified to publish a case in which the unprotected brain was treated with large doses of roentgen rays, and in which the authors were in a position to examine the brain histologically.

CASE REPORT

The patient was a female, aged thirty-four. At the age of seven months she fell into a fire and suffered severe burns of the right hand and the right side of the scalp; the former healed with scar contraction of the palm; the latter healed slowly with a dense adherent scar, leaving a small area in the center which did not heal even in the following years. This area drained more and more, and finally increased in size causing local pain as well as generalized headache.

In September, 1936, the patient was examined in the Out-Patient Department, and a biopsy of the ulcerated area revealed "squamous cell carcinoma, Grade 1, radioresistant." The patient was given roentgen therapy. On December 2, 1936, 1,500 r, focal skin distance 12 inches, filter 1 mm. Al, 150 kv. (peak), 5 ma., 20 minutes; March 26, 1937, 2,160 r, focal skin distance and filter as above, 98 kv. (peak), 5 ma., 3 inch cone, 20 minutes. The patient did not come again for examination until August 19, 1937. Roentgen examination revealed a bone destruction, particularly in the outer table below the skin cancer, in its appearance an inflammatory process rather than a malignant infiltration, but there might have been an element of both. The skin tumor and the underlying bone were removed by excision and curettement, and the pathological report (September 28, 1937) revealed a squamous cell epithelioma of the wall, Grade 1, radioresistant, with not absolutely certain infiltration of the bone.

On October 2, 1937, the following roentgen treatment was given: 1,080 r, no filter, focal skin distance 12 inches, 3 inch cone, 5 ma., 98 kv. (peak), 10 minutes; this treatment was repeated on November 5, 1937 (total 2,160 r).

On December 20, 1937, the destruction of the skull was seen to have progressed in the center,

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destroying both tables of the skull. The patient again disappeared for some months, and during this time the process spread progressively. March 15, 1938, roentgen treatment, applied tangentially anteriorly, 2,000 r, focal skin distance 12 inches, 98 kv. (peak), 5 ma., 3 inch cone, no filter; on March 19, 1938, this treatment was repeated, this time posteriorly (4,000 r total).

On July 25, 1938, no metastases could be observed, there were no neurological signs, and the blood count revealed 3,640,000 erythrocytes, 8,500 leukocytes, 78 per cent hemoglobin. On July 30, the tumor, as well as the underlying bone (3 cm. beyond the neoplastic tissue), was removed. The dura, not adherent, appeared considerably thickened and showed an inflammatory reaction corresponding to the center of the lesion. Postoperatively there were no signs of cerebral injury or increased intracranial pressure. During the period of recovery, when a fair granulation was produced by wet azochloramide dressings 1:3,300, a dura fistula was discovered. Oh September 2, 1938, the thickened necrotic portion of the dura was excised revealing its under surface to be perfectly smooth and not adherent to the brain. A superficial small cortical abscess was easily evacuated, and an iodoform pack inserted.

Whereas there were no signs in the central nervous system during the first few days after removal of the abscess, there was a slight progression of the abscess cavity, and on September 12 the left upper extremity was spastic paraplegic, the left lower weak with exaggerated reflexes. Abdominal reflex absent, no sensory disturbances. The abscess cavity then grew smaller, and on September 26 there was no drainage. However, a cerebral fungus developed with considerable sloughing of the protruding portion. The neurological findings remained unchanged.

After a Thiersch graft on September 30 the cerebral hernia receded, and had disappeared by December 1, 1938. The motor power of the lower extremity returned, and the patient was able to walk. The biopsy of the dura showed an inflammatory process as well as replacement of the dura tissue by neoplastic cells; the diagnosis was epithelioma.

The patient left the hospital but returned in January, 1939. Biopsy on January 10 revealed a diffuse recurrence of the neoplasm. On January 28, irradiation to the exposed part of the brain and dura, 85 kv. (peak), 5 ma., no filter, focal skin distance 20 inches, 3 inch cone, 650 r; repeated on February 3 (1,300 r total). Very marked reaction followed: cerebral hernia, 10–12 cm. diameter, projecting about 5–6 cm. above the level of the scalp.

On April 14, 1939, the patient was re-admitted to the hospital with severe headache, ulcerations along the anterior border of the original line of excision, left hemiplegia with exaggerated tendon reflex, and slight diminution of pain sensation. The cerebral hernia receded slowly in the next month when considerable sloughing, necrotic cortical tissue was cut off. On July 5, radium plaques were applied, five tubes, each containing 10 mg. of radium (1,400 mg-hr. to the anteromedial border); on July 12, 1,862 mg-hr. to the anterior border. No satisfactory effect.

While the process then receded in the center, there was an extension along the margins. Therefore another operation was performed. In spite of severe hemorrhages during the operation the patient recovered, and after a few weeks healthy granulations appeared. But by the middle of September the wound ceased to show further signs of healing, and the entire operative site presented irregular, elevated areas of pale granular tissue rising above the level of the underlying firmer granulation tissue. On September 29, biopsies were taken from five widely scattered areas and all of these showed residual islands of neoplastic cells with a tendency to formation of epithelial pearls in some areas. From this point on the patient experienced severe headache requiring repeated lumbar punctures and heavy sedation. She remained perfectly conscious and oriented until a few days before her death when she showed some evidence of confusion and disorientation. No further neurological signs developed during this time, however. The patient began to run an irregular temperature on December 17. On December 26 she lapsed into a coma and died on December 27, 1939.

Summary. Roentgen resistant squamous cell carcinoma of the skull with destruction of the bones and infiltration of the dura which was partly removed. Purulent infection of the wound with abscess formation occurred, followed by a left hemiplegia accompanied by protrusion of the brain and persisting until death.

Treatment. Roentgen therapy: December, 1936, 1,500 r; March, 1937, 2,160 r; October,

1937, 1,080 r; March, 1938, twice 2,000 r (open skull); January 28 and February 3, 1939, 650 r each. Finally radium, 1,400 mg-hr. anteromedial border (July 5, 1939) and 1,862 mg-hr. (July 12, 1939).

Postmortem Examination. Histopathological findings: The brain is partly covered with a thick connective tissue membrane replacing the respective part of the dura mater. This membrane consists of a matrix of cell-poor reticulin

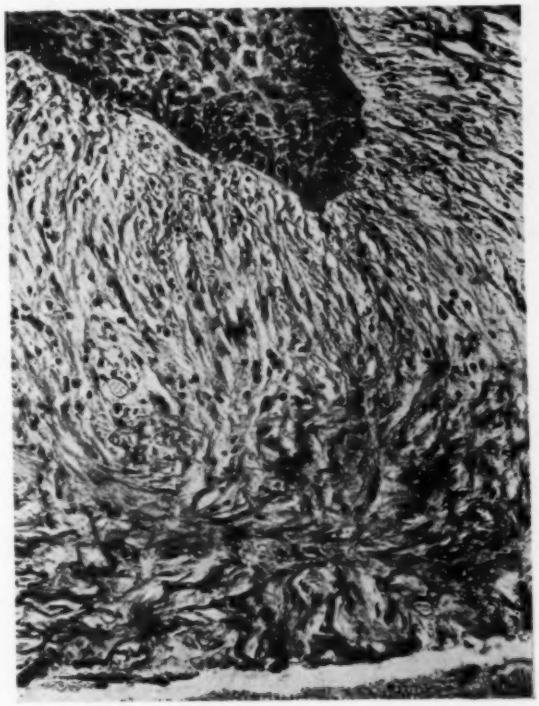

Fig. 1. Cicatricial dura mater.

from which many nucleated fibrils run toward the nodes and cones of the cancer, surrounding it with a chain of cells and a fine membrane (Fig. 1). Near this cicatrix the dura mater shows an intrusion of cancer cells via perivascular spaces (Fig. 2), forming small cell chains and bands throughout the dura. The connective tissue of the dura is spongy, rather loose, and edematous. Occasionally one finds some cancer pearls and near them single cancer cells (Fig. 3) with a division of the nuclei indicating the progression of the process. Unfortunately a suppurative process has changed the picture so that one encounters in addition to the cancer, a purulent infiltration of the dura (Fig. 3).

Fig. 2. Dura mater. Cancer cells along the blood vessels.

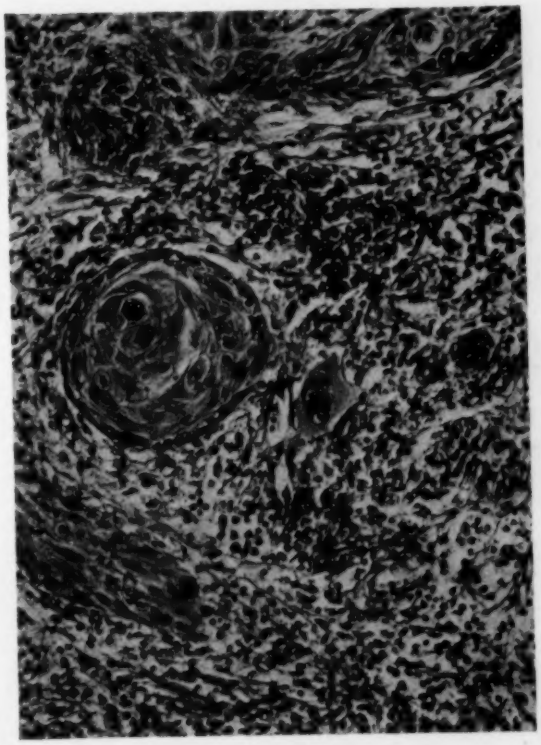

Fig. 3. Cancer node and inflammation within the dura mater.

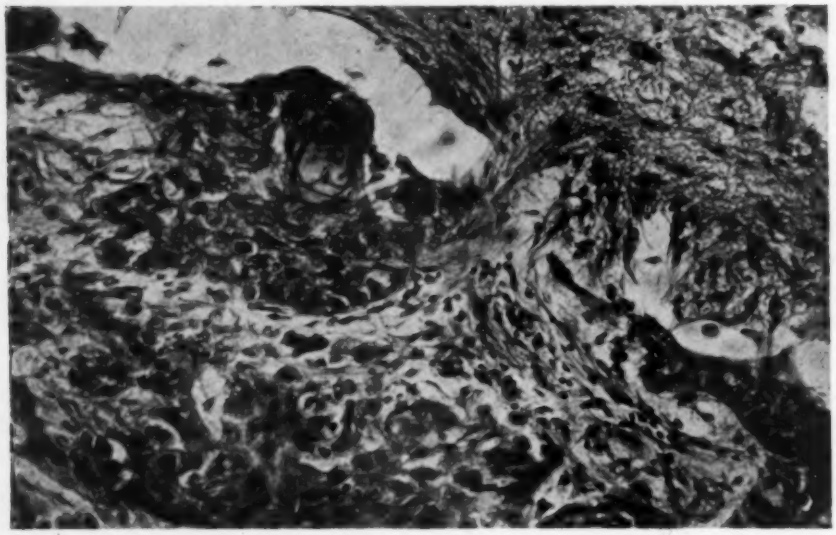

FIG. 4. Pia mater. Cortical glia intruding into the pia.

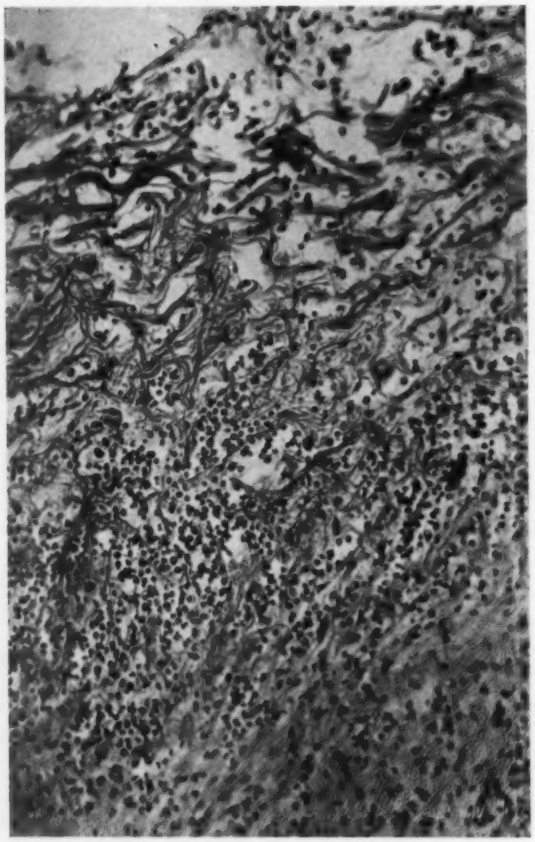

FIG. 5. Inflammation of pia mater and cerebral cortex.

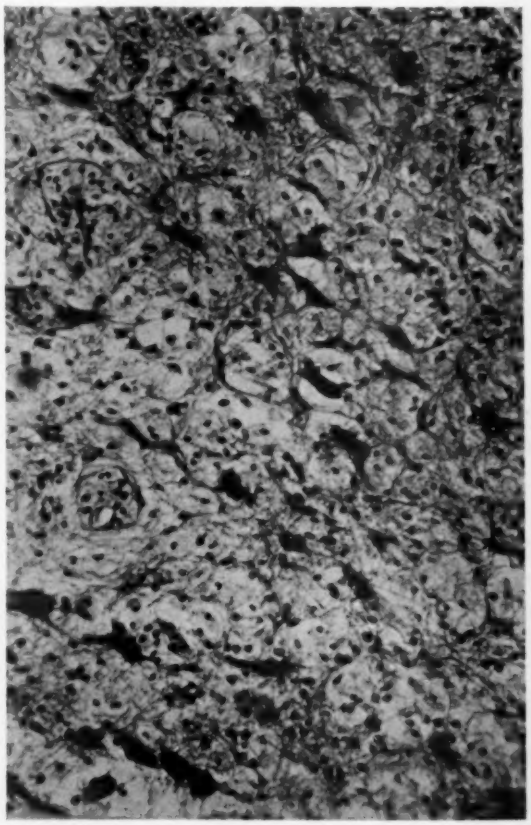

FIG. 6. Malacia and giant reactive glia cells (cortex).

The pia mater is also filled with cancer cones and nodes which, however, do not spread into the brain. There is a space between pia and brain which contains glia fibrils entering into the pia (Fig. 4).

In the brain, three different pictures can be observed: (1) In one part there is suppurative encephalitis (Fig. 5). (2) Another part shows multiple malacias. One sees here compound granular cells around small blood vessels the walls of which are homogeneous. Groups of these cells are surrounded by giant astrocytes (Fig. 6) which adjust their form to the circularly arranged compound granular cells. There is some beginning fibrillogenesis (Fig. 7). Another group of these giant glia cells is disintegrated, with vacuoles and clasmatodendrosis (ameboidosis) (Fig. 8). (3) Along the pial blood vessels tumor cells infiltrate into the brain and isolate parts of the cortex that are completely necrotic and present only a glia fibril network with some shrunken glia cells (Fig. 9).

The transition from these affected portions to the normal areas is represented by a stripe of edematous tissue with wide gaps. This tissue shows only little reaction in general, which is easily explained by the obstruction of the perivascular lymph spaces by compound granular or cancer cells.

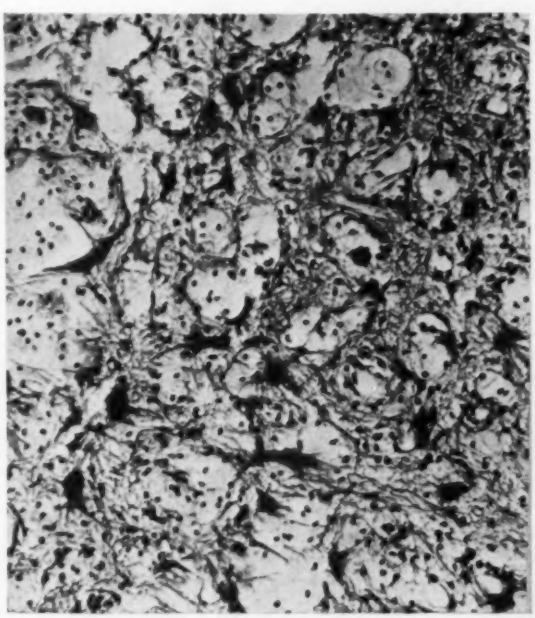

Fig. 7. Fibrillogenesis of the giant glia cells.

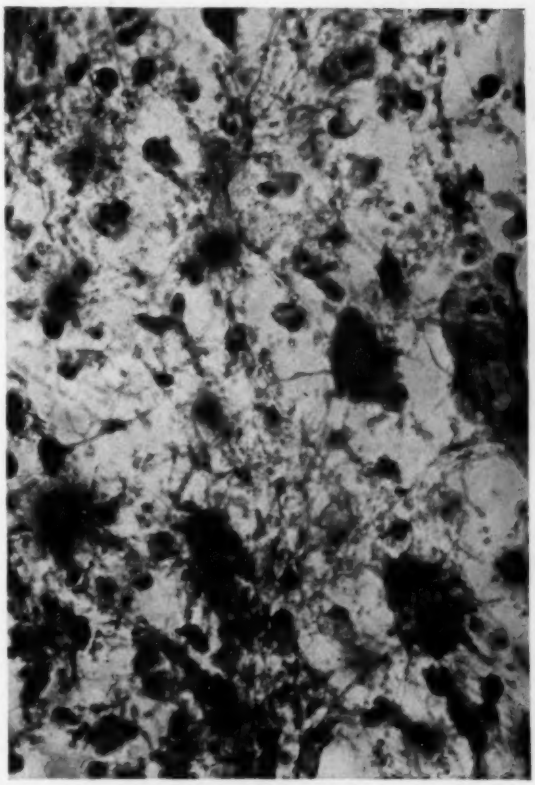

Fig. 8. Ameboidism of the glia cells.

The destruction of the ganglion cells near areas of necrosis or malacia is very advanced. The cells are shrunken, dark without a vestige of a nucleus, and present no dendrites. These changes occur, however, only rather close to the foci. Next there is seen a thin stripe of swollen ganglion cells, and finally normal cells. The damage of the most severely affected portions of the cortex extends into the white matter.

The changes in the blood vessels vary according to the size of the vessels. The capillaries present swollen intima cells, their walls are occasionally pale, their lumina are obstructed. In the larger vessels the intima is not swollen, the walls are rather homogeneous, the nuclei of the muscularis and the adventitia are not recognizable, and the homogenization of the media resembles that in arteriosclerosis. Occasionally a small artery is stained so dark in hemalum and eosin preparations that calcification is suggested. The adventitia is pale and consists of a loose network of fibrils without nuclei.

Macroscopically the brain gave the impres-

sion of hemiatrophy of the affected hemisphere. Accurate examination, however, revealed that the shape of this hemisphere was changed because the cicatrix below the wound had shrunk and had shifted the entire ventral part of the hemisphere ventrally and laterally.

The examination of the contralateral side showed relatively normal conditions. Unfortunately there was a generalized shrinkage; some of the pyramidal cells were pyknotic, homogeneous and black stained, but their nuclei were normal. Most of the cells presented a normal staining in Nissl preparations. Hemalum and eosin stained sections failed to show any abnormality.

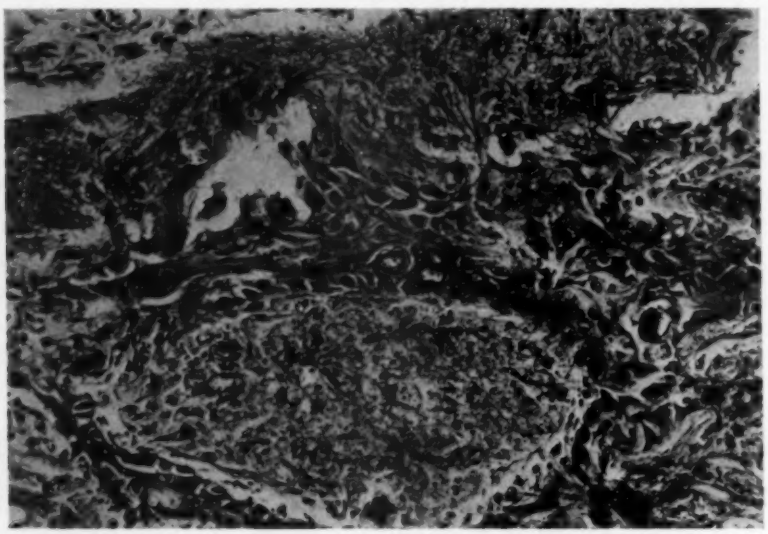

FIG. 9. Pia mater and cortex. Infiltration of cancer cells.

The question arises as to which of the changes are caused by cancer and inflammation, and which by the irradiation. That is difficult to decide in the present case because the changes caused by cancer, and those caused by the irradiation are partly the same, namely obstruction of blood vessels with its sequelae. In some regions, however, in the present case the pia mater is not affected by cancer, and yet these regions present changes as described in the experiments of Davidoff, Dyke, Elsberg and Tarlov. Their findings are in agreement with those of W. Scholz who, in animals surviving for a longer time, found hemorrhages, malacias, and progressive as well as regressive changes of the glia accompanied by progressively deteriorating clinical signs. Whereas Mogilnitzky and Podljaschuk particularly stressed the changed permeability of the blood vessel walls, Scholz claimed that the damage of the blood vessel walls leads to roentgen changes of the brain resembling those described by Windholz. Scholz in addition assumed a functional lesion of the vasomotor nerves explaining the progressive clinical signs.

In the present case the changes are not in complete agreement with those described by Scholz,[12] and by Lyman, Kupalov and Scholz.[5] The adventitia appears rather pale in hemalum and eosin preparations without the hyalin transformation seen in Scholz's[12] experiments, and the fibrils are distended by deposition of compound granular cells. Some homogenization of the media is seen resembling an incipient calcification.

The most striking fact in the present case was the relatively late onset of the clinical signs. Six months after the irradiation of the open brain, hemiplegia occurred which corresponds with Scholz's experimental

statements. It is evident that neither the abscess nor cancer was the cause of this hemiplegia since both were removed two months before the onset of paresis when examination revealed that the dura was not adherent and that there were no signs of cerebral injury. In addition, the freshness of the malacias suggests with some reservation that the irradiation caused the damage as well as the clinical signs.

All the writers stress the reaction of the glia, particularly the progressive and regressive changes occurring close to each other. The same is seen in irradiation gliomas as described by Marburg,[6] Roussy, Laborde and Lévy,[11] Tarlov,[13] Dyke and Davidoff,[2] and many others. As mentioned, this reaction surpasses the usual glia reaction seen in secondary degeneration, and on the other hand, neuronophagia is absent in spite of a degeneration, though not very extensive, of the ganglion cells. That means that the trophic and defensive reaction of the glia is well preserved, or may be improved by irradiation. The ameboidism observed at the same time is no proof to the contrary since according to Pollak[10] progressively changed glia cells when assuming trophic functions are transformed into compound granular cells (dysplasia).

As for the ganglion cells, it was not possible in the present case to find vacuolization as described by Davidoff, Dyke, Elsberg and Tarlov.[1] Nor were there such pale, atrophic ganglion cells as described by Spielmeyer as sequelae of anemia. Near the foci there were shrunken, malformed, homogeneous cells, stained dark with Nissl methylene blue (coagulation necrosis according to Marburg), without the vestige of a nucleus. In their neighborhood some swollen ganglion cells resembled those in acute degeneration. A transition from one state to the other could not be demonstrated. In any case, the changes were seen only in close proximity to the malacic or necrotic foci which did not extend deeper than into the white matter of the cortex convolution; that means that the effect of the irradiation ended 0.5 cm. from the surface. The intactness of the other parts of the cortex as far as they were examined is surprising.

SUMMARY AND CONCLUSIONS

In a case with a roentgen resistant squamous cell carcinoma of the skull, the bone was perforated by the cancer. The brain, already previously irradiated, now lying free by the perforation, was treated with roentgen radiation (4,000 and later 1,300 r) and finally with radium.

In spite of the infiltration of the cancer into the brain, and a concomitant abscess, the changes which occurred may be attributed in part to the irradiation because of their resemblance to experimental findings.

The primary changes are those of the blood vessels. The obstruction of the capillaries, the disintegration, and the increased permeability of the blood vessel walls may also be due to a functional impairment of the vasomotor nerves.

A vivid (defensive and atrophic) glia reaction as well as a particular cell degeneration in the neighborhood of the malacic foci are to be stressed. The relative intactness of the contralateral side is striking.

REFERENCES

1. Davidoff, L. M., Dyke, C. G., Elsberg, C. A., and Tarlov, I. M. Effect of radiation applied directly to brain and spinal cord; experimental investigations on Macacus rhesus monkeys. *Radiology*, 1938, *31*, 451–463.
2. Dyke, C. G., and Davidoff, L. M. Roentgen Treatment of Diseases of the Nervous System. Lea & Febiger, Philadelphia, 1942.
3. Elsberg, C. A., Davidoff, L. M., and Dyke, C. G. Roentgen treatment of tumors of brain or spinal cord in operating room by direct radiation through the open wound. *Surg., Gynec. & Obst.*, 1936, *63*, 794–796.
4. Elsberg, C. A., Davidoff, L. M., and Dyke, C. G. Roentgen treatment of tumors of brain in operating room by direct radiation through the open wound. *Bull. Neurol. Inst. New York*, 1937, *6*, 19–32.
5. Lyman, R. S., Kupalov, P. S., and Scholz, W. Effect of roentgen rays on central nervous sys-

tem; results of large doses on brains of adult dogs. *Arch. Neurol. & Psychiat.*, 1933, *29*, 56–87.

6. MARBURG, O. Pathologische Untersuchungen über die Einwirkung der Röntgenstrahlen auf Hirntumoren, zugleich ein Beitrag zur Histologie des Glioms. *Arb. a. d. neurol. Inst. a. d. Wien. Univ.*, 1928, *30*, 171–184.
7. MARBURG, O., and SGALITZER, M. Die Röntgenbehandlung der Nervenkrankheiten. Sonderbände zur Strahlentherapie. Band XV. Hans Meyer, Editor. Urban und Schwarzenberg, Berlin, 1930.
8. MOGILNITZKY, B. N., and PODLJASCHUK, L. D. Zur Frage über die Wirkung der Röntgenstrahlen auf das zentrale Nervensystem. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1929, *40*, 1096–1108.
9. MOGILNITZKY, B. N., and PODLJASCHUK, L. D. Röntgenstrahlen und sogenannte "hämatoenzephalische Barriere." *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1930, *41*, 66–75.
10. POLLAK, E. Studien zur Pathologie der Neuroglia. *Arb. a. d. neurol. Inst. a. d. Wien. Univ.*, 1919, *22*, 296.
11. ROUSSY, LABORDE, and LÉVY. Traitement des tumeurs cérébrales par la radiothérapie. *Rev. neurol.*, 1924, *2*, 129.
12. SCHOLZ, W. Experimentelle Untersuchungen über die Einwirkung der Röntgenstrahlen auf das reife Gehirn. *Ztschr. f. d. ges Neurol. u. Psychiat.*, 1934, *150*, 765–785.
13. TARLOV, I. M. Effect of roentgenotherapy on gliomas. *Arch. Neurol. & Psychiat.*, 1937, *38*, 513–536.
14. WINDHOLZ, F. Zur Kenntnis der Blutgefässveränderungen im röntgenbestrahlten Gewebe. *Strahlentherapie*, 1937, *59*, 662–670.

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EDITORIALS

FACTITIAL REACTIONS IN CONNECTION WITH IRRADIATION OF THE PELVIS

THE successful irradiation of most deep-seated malignant neoplasms necessitates the administration of such large doses that a certain effect on the surrounding normal structures often cannot be avoided. The clinical course and pathological significance of this effect have formed the topics of numerous studies, and there is now a voluminous literature published on the subject.

Of late, however, in not a few of the articles a rather disproportionate emphasis is laid on the injurious nature of the irradiation effect. This applies especially in regard to the pelvis, where such highly sensitive organs as the lower portions of the intestinal and urinary tracts are included within the irradiated volumes. A brief review of the entire subject in the light of the newer developments may therefore not appear out of place.

In 1930, Buie and Malmgren[1] used the term "factitial proctitis" in order to designate pathologic phenomena observed within the rectum following radium or a combination of radium and roentgen therapy given for extrarectal lesions. The generalization of this term to include all reactions and sequelae which are noted in organs or structures other than those for which the irradiation was intended may serve a good purpose. Thus one could speak of factitial colitis, cystitis, late ulceration, and so forth, giving expression to the fact that the process was unintentionally produced but that it could not be avoided. The adoption of such nomenclature might, with time, perhaps lead to the elimination of the injudiciously used term of "burn" which creates considerable confusion and distrust. Obviously, cases are encountered in which the irradiation damage is the result of a gross overdosage or a faulty technique but our interest here concerns those instances in which the reactions and late sequelae follow more or less standard techniques as employed in current radiologic practice.

Factitial Reactions of the Lower Intestinal Tract. In irradiating the pelvis, a reaction may develop in that portion of the intestinal tract which normally is situated in the pelvis or which, under certain abnormal conditions, may drop or be pulled into the pelvis, as for example, by adhesions. The parts involved are usually the rectum, the rectosigmoidal junction, the loops of the sigmoid and occasionally the loops of the small intestine. In a decreasing order of frequency and severity the reactions are observed in the treatment of cancer of the vaginal canal, prostate, cervix uteri, corpus uteri, bladder, ovaries and certain less common lesions. At times they are the result of roentgen therapy alone, especially when the fractionated protracted method is used over a long period, but more often they are due to the effect of radium, or a combination of roentgen therapy and radium.

There were a number of articles published during the last few years dealing with all effects of the factitial reactions of the lower intestinal tract. Corscaden, Kasabach and Lenz[2] Todd,[3] Aldridge,[4]

[1] Buie, L. A., and Malmgren, G. E. Factitial proctitis; a justifiable lesion observed in patients following irradiation. *Internat. Clin.*, 1930, *3*, 68–77.

[2] Corscaden, J. A., Kasabach, H. H., and Lenz, M. Intestinal

Randall and Buie,[5] and others, have furnished us with a very vivid description of the clinical and roentgen appearance of such conditions. It is clear from these descriptions that we are dealing with an acute and chronic phase of the process. The acute phase starts toward the end or immediately after the completion of the irradiation series and is of a transitory nature. Clinically, it manifests itself by tenesmus, frequency of defecation and occasional rectal bleeding. Proctoscopic or sigmoiscopic examination reveals diffuse hyperemia of the rectal mucosa and the presence of fibrinous flakes, usually more pronounced in the areas near where the radium was applied. This mucositis often assumes a thick edematous or granular appearance and is followed by a mucosal desquamation similar to that observed in mucosal membranes of other locations or in the teguments of the skin. A roentgen study by means of a barium enema shows practically no findings of significance during this phase. The acute mucositis in the great majority of cases clears up completely without further after effect. In some instances, however, it gives place to an atrophy and telangiectasis of the mucosa which then are permanent manifestations forming the source of recurrent bleedings or local bowel symptoms of varying degree.

The chronic phase is characterized by more deep-seated involvement of the structures. The various authors place the onset at an average of six to ten months after treatment, with a minimum interval of one week and a maximum interval of ten years or longer. The first warning that anything is wrong occurs when the patient suddenly develops a gross rectal hemorrhage. This is followed by discomfort on defecation, later by pain and eventual stenosis. All investigators stress the fact that at this time the proctosigmoidoscopic as well as the roentgen appearance is that of a pseudocarcinoma and that biopsy is often necessary to make a differential diagnosis. On inspection there is a deep grayish-white ulcer in the center of a proliferating granulation tissue and not infrequently there is a massive pelvic induration leading to a frozen pelvis. It is probable, however, that in this latter instance a residual infection from the original sphacelated malignant neoplasm accounts to no small degree for the deep induration. On roentgen study filling defects, tubular constrictions and rarely complete stenosis form the chief manifestations. In occasional cases communicating fistulae may be encountered.

A review of the literature reveals considerable discrepancy concerning the incidence of the acute phase of the factitial lower bowel reaction but there is good agreement concerning the late sequelae. This is understandable. In the great majority of the acute reactions no systematic observations are made and, since they are of a transitory nature, most of them pass unnoticed. In some institutions there is a tendency to consider the presence of an acute mucositis of the lower bowel and bladder as an indicator of proper dosage for the irradiation of certain well chosen pelvic lesions. Under such circumstances the incidence may be almost 100 per cent. For the late sequelae, Buie and Malmgren,[1] noted a percentage incidence of 3.13 in 2,073 cases treated at The Mayo Clinic from 1921 to 1930, occurring mostly following roentgen and radium therapy of carcinoma of the cervix and fundus uteri, carcinoma of the ovary and other pelvic lesions. Later, when the dose was considerably increased, Corscaden, Kasabach and Lenz[2] reported an incidence of 8.7 per cent in 442 cases of uterine carcinoma. Todd,[3] in analyzing the cases irradiated at the Christie Hospital and Holt Radium Institute of Manchester found a percentage incidence

injuries after radium and roentgen treatment of carcinoma of the cervix. Am. J. Roentgenol. & Rad. Therapy, 1938, *39*, 871–887.

[3] Todd, T. F. Rectal ulceration following irradiation treatment of carcinoma of the cervix uteri; pseudo-carcinoma of the rectum *Surg., Gynec. & Obst.*, 1938, *67*, 617–631.

[4] Aldridge, A. H. Intestinal injuries resulting from irradiation treatment of uterine carcinoma. *Am. J. Obst. & Gynec.*, 1942, *44*, 833–857.

[5] Randall, L. M., and Buie, L. A. Factitial proctitis. *Am. J. Obst. & Gynec.*, 1943, *45*, 505–512.

of 4.5 of all treated cases. He noted a similar incidence at the Marie Curie Hospital of London and from a personal visit to Radiumhemmet of Stockholm gathered the impression that this was also approximately the incidence in Heyman's cases. Chydenius,[6] in using the "Stockholm method" of irradiation, found a percentage incidence of 4.8 in 321 cases of carcinoma of the cervix uteri, occurring mostly in the very advanced cases. Aldridge[4] in 1942 published a series of 189 cases of uterine carcinoma treated by irradiation, in 16.9 per cent of which intestinal injuries occurred, but this high incidence, as the author states, is due to the fact that every case of proved injury has been reported even if the findings were extremely mild, giving rise to only transient symptoms. In a more recent publication, McIntosh and Hutton,[7] dealing with the same material from a different point of view and adding some additional cases, likewise express the opinion that a number of these cases would not have been discovered had the authors not been particularly engaged in looking for them.

The possibility of factitial intestinal injuries being generally admitted, attempts were made to devise procedures which would prevent, or at least minimize, their occurrence. Aldridge[4] compiled a rather long list of suggestions, some dealing with the technical application of the radium so that it would not get dislodged or lie too close to sensitive mucosal membranes, others advocating various studies, including even a pre-irradiation exploratory laparotomy to determine whether loops of intestine are adherent in portions that might subject them to too much irradiation. The most important suggestion, however, refers to a change in the actual technique of irradiation. It was thought that by decreasing the amount of radium and roentgen radiation that is given at one time and by spacing it over a longer period the difficulty could be overcome, at least to a certain extent. Such a procedure has been adopted in a number of the leading institutions during the past eight or ten years. Yet, Cosbie,[8] in analyzing the cases of bowel injury at the Ontario Institute of Radiotherapy of Toronto, where the change to the newer technique was made in 1937, states that as a result of the fractionation of the radium and the introduction of higher voltage roentgen rays, proctitis was less common, but late ulcerations became more frequent. A very valuable article dealing with a multiplicity of factors aiming to eliminate irradiation injuries in the treatment of cancer of the cervix was published not very long ago by Charles L. Martin.[9]

As far as the treatment of the late sequelae of the bowel is concerned, there is now a unanimity of opinion that this must be along conservative lines. Actual or chemical cauterants should never be used and the surgical procedures should be reserved for carefully chosen cases. This non-operative treatment was recently stressed by McIntosh and Hutton[7] who found that even severe and extensive bowel injuries often heal with very little functional impairment, though marked anatomic defects may be present. There are a few cases of fatal outcome recorded in the literature.[8]

Factitial Cystitis. What has been said about the factitial irradiation effect on the lower intestinal tract also applies to the bladder. The evolution and clinical course of the mucositis follows the same pattern and the mechanism of the late sequelae is based on the same principles.

There are a few distinguishing features. One of these is the fact that the bladder mucosa is slightly less radiosensitive than the bowel mucosa. As a result, the symptoms in the acute phase are somewhat less

[6] Chydenius, J. J. Strahlenschäden des Darmes und der Harnblase bei der Radiumbehandlung des Carcinoma colli uteri. *Acta radiol.*, 1942, *23*, 1–7.

[7] McIntosh, Harriet C., and Hutton, J. E. Clinical and roentgen aspects of irradiation stricture of the rectum and sigmoid. AM. J. ROENTGENOL. & RAD. THERAPY, 1944, *52*, 647–662.

[8] Cosbie, W. G. Complications of irradiation treatment of carcinoma of the cervix. *Am. J. Obst. & Gynec.*, 1941, *42*, 1003–1008.

[9] Martin, C. L. Elimination of irradiation injuries in the treatment of cancer of the cervix. AM. J. ROENTGENOL. & RAD. THERAPY, 1943, *49*, 494–503.

pronounced and the appearance of the chronic ulcers occurs at a later date, the average being about two years and six months after irradiation. For the same reason, the incidence of the late sequelae is smaller. Dean and Slaughter,[10] for example, in analyzing a group of 3,041 patients treated with irradiation for uterine and ovarian diseases at the Memorial Hospital of New York, found that only 2.48 per cent of these patients sought relief later because of injured bladders. The incidence varied with the disease irradiated. It was least for fibromyoma (0.38 per cent) and greatest for postoperative recurrent carcinoma of the cervix (7.51 per cent).

Another point is that healing of a factitial bladder ulceration is dependent on the size and location of the ulceration. In this respect the situation is similar to that observed in primary ulceration of the bladder when interstitial radium is applied alone or in conjunction with roentgen therapy for the treatment of a neoplasm of the bladder proper. Herger and Sauer,[11] in analyzing a group of 279 patients with tumors of the bladder treated with radon implants, found that in no less than 80.7 per cent of the cases extensive ulceration resulted. In those cases in which the ulcerations eventually healed, it took an average of 10.6 months for the healing process to be completed. In a significant number of cases the ulceration never healed, especially if they were larger than 5 cm. in diameter. As a rule, the duration of the healing was in direct proportion to the size of the area involved and the amount of interstitial radium used. Ulcerations of the loose walls of the bladder healed faster than those in the fixed parts of the bladder. The development of incrustations prolonged the duration of the healing.

[10] Dean, A. L., and Slaughter, D. P. Bladder injury subsequent to irradiation of the uterus. *J. Urol.*, 1941, *46*, 917–924.

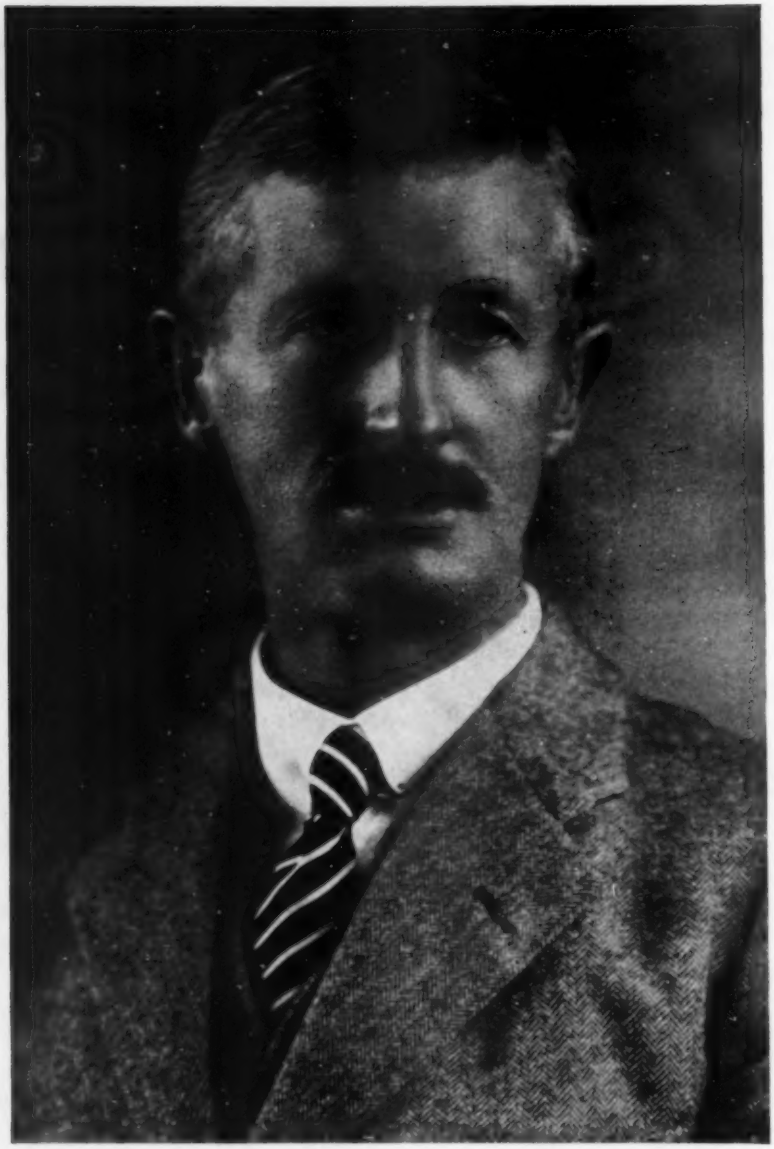
[11] Herger, C. C., and Sauer, H. R. Occurrence and clinical course of radium reactions following use of radon implants in treatment of carcinoma of bladder. *J. Urol.*, 1942, *47*, 141–147.

Finally, a third point has to do with the peculiar anatomy of the bladder. Hueper and his coworkers[12] recently produced experimental radiocystitis in a series of 22 male mongrel dogs and observed the changes up to a period of eleven months. They noted all the pathologic lesions equivalent to those found in radiocystitis of the human. But, surprisingly, a secondary infection of the bladder with fecal flora also developed. The authors attributed this to the irradiation injury of the rectovesical septum and the vesical mucosa. This observation is important since it may help to explain why the administration of sulfa drugs in the acute phase of factitial or actual radiocystitis in the human produces such prompt relief of symptoms. Even in cases of late sequelae rapid improvement is obtained as soon as the infection is eliminated.

All in all, it appears that with the heavy irradiation, amounting to several erythema doses at many critical points, as is practiced nowadays in the treatment of malignant neoplasms situated within the pelvis, factitial reactions cannot be avoided. Acute mucositis of the lower bowel and bladder represents a common manifestation and its degree often serves as a control of the correctness of the dosage applied. Late sequelae of the lower intestinal tract are encountered in about 5 per cent and of the bladder in about 3.5 per cent of all treated cases. As Randall and Buie[5] say, they represent ordinary risks that the patient assumes when such treatment is undertaken. With very few exceptions, their ultimate prognosis is good and in comparison with the original lesion for which the radiation therapy was given, the inconveniences they cause to the patient are disproportionately small.

T. Leucutia

[12] Hueper, W. C., Fisher, C. Virginia, de Carvajal-Forero, J., and Thompson, M. R. Pathology of experimental roentgencystitis in dogs. *J. Urol.*, 1942, *47*, 156–167.



ALEXANDER HOWARD PIRIE, M.D., F.R.S.M. (LONDON), F.F.R.

1875-1944

DR. ALEXANDER HOWARD PIRIE, Past President and Life Member of the American Roentgen Ray Society, long-time (1911–1938) Radiologist-in-Chief, Royal Victoria Hospital, Montreal, and Lecturer in Radiology, Faculty of Medicine, McGill University, died in the Ross Pavilion of the Royal Victoria Hospital on November 23, 1944, after a sudden and short illness near the close of his sixty-ninth year.

Son of a physician of Dundee, Scotland,

young "Sandy" Pirie was completing his education at St. Andrew's and Edinburgh University when Roentgen astounded the scientific world with the announcement of his discovery. Together with his brother, who was to become one of the early martyrs to overexposure, Dr. Pirie soon became absorbed in the medical application of the x-rays. Following a period of study at the Sorbonne, and a visit to Canada in 1897, he became established in Harley Street, London, subsequently becoming associated with another pioneer in British Radiology, the late noted Dr. Robert Knox.

In 1911, relinquishing appointments at St. Bartholomew's, the Prince of Wales and Mount Vernon Hospitals, Dr. Pirie accepted the invitation of the Governors of the Royal Victoria Hospital, Montreal, to become its Radiologist-in-Chief, succeeding the late Professor Girdwood. His versatile ingenuity there enjoyed full play in the adoption and adaptation of each new development in equipment for diagnosis and treatment. The Department of Radiology of this eminent Canadian hospital enjoyed progressive growth and enlargement under his direction.

Having been Medical Officer to the London Scottish Regiment before coming to Canada, Major Pirie served with distinction during the first World War as Radiologist of the McGill General Hospital Unit, Canadian Expeditionary Force. During his service in France, he developed a resonance apparatus for the location of metallic foreign bodies.

His public service continued as a member of the Advisory Committee on Radiology, National Research Council of Canada, until 1938; one of the consultants to the Department of Pensions and National Health in Montreal and its hospital at Ste. Anne de Bellevue and as Mayor of his residential village, Baie d'Urfe, until shortly before his death.

The members of the American Roentgen Ray Society will long remember him for his keen scientific interest and comment on all technical and interpretive developments, his "roentgenometer" for gauging the degree of accommodation of the eyes for the roentgenoscope, the demonstration of the fact that the retina is sensitive to roentgen rays—for which he was awarded the Society's Gold Medal in 1932—and especially the traditional singing of "Alouette," which "Sandy" led at the annual banquets.

His penchant for and curiosity in all the natural sciences was manifest by an active interest in ornithology and astronomy, the development of extensive gardens wherein he was wont to propagate special plants from seeds produced by selected flowers of the year before, "soil-less" culture of flowers, vegetables and shrubs brought from his Florida home to his "habitant" suburban home on Lac St. Louis and the recording of some of his impressions in wood-carvings.

These interests were enjoyed with him by Ethel Martin, daughter of an Ontario physician and an authority on the birds of eastern Canada, whom he married in 1903, but who died in 1938 after a prolonged illness, and the second Mrs. Pirie, Mary C. Sayer, whom he married late in 1939. Only three months before his death, the Piries had occupied their new country estate at Alstonvale on the Ottawa River, and had transplanted many of their flowers to bloom there.

Member of the American Roentgen Ray Society since 1912, President in 1927, and Life Member since 1943; member of the British Institute of Radiology; Fellow of the Royal Society of Medicine (London) and the British Faculty of Radiologists; one of the charter members of the Canadian Association of Radiologists; Canadian delegate to the International Congress of Radiology (London, 1925 and Stockholm, 1928), Dr. Pirie contributed much to the development of radiology, especially in Canada.

He was a sincere and loyal friend, who will be greatly missed by his colleagues.

Carleton B. Peirce

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this section solicited promptly after the events to which they refer.

MEETINGS OF ROENTGEN SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. H. Dabney Kerr, University Hospital, Iowa City, Iowa. Annual meeting: Netherland Plaza Hotel, Cincinnati, Ohio, Sept. 18–21, 1945.

AMERICAN COLLEGE OF RADIOLOGY
Secretary, Mac F. Cahal, 540 N. Michigan Ave., Chicago.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. U. V. Portmann, Cleveland Clinic, Cleveland, Ohio.

ARKANSAS RADIOLOGICAL SOCIETY
Secretary, Dr. J. S. Wilson, Mack Wilson Hospital, Monticello, Ark. Meets every three months and also at time and place of State Medical Association.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y. Annual meeting: 1945, to be announced.

RADIOLOGICAL SECTION, BALTIMORE MEDICAL SOCIETY
Secretary, Dr. Walter L. Kilby, Baltimore. Meets third Tuesday each month, September to May.

SECTION ON RADIOLOGY, CALIFORNIA MEDICAL ASSOCIATION
Secretary, Dr. Earl R. Miller, University of California Hospital, San Francisco, Calif.

RADIOLOGICAL SECTION, CONNECTICUT MEDICAL SOCIETY
Secretary, Dr. Max Climan, 242 Trumbull St., Hartford, Conn. Meets bi-monthly on second Thursday, at place selected by Secretary. Annual meeting in May.

SECTION ON RADIOLOGY, ILLINOIS STATE MEDICAL SOCIETY
Secretary, Dr. H. W. Ackemann, 321 W. State St., Rockford, Ill.

RADIOLOGICAL SECTION, LOS ANGELES COUNTY MEDICAL ASSOCIATION
Secretary, Dr. Roy W. Johnson, 1407 S. Hope St., Los Angeles, Calif. Meets on second Wednesday of each month at the County Society Building.

RADIOLOGICAL SECTION, SOUTHERN MEDICAL ASSOCIATION
Secretary, Dr. Roy G. Giles, Temple, Texas.

BROOKLYN ROENTGEN RAY SOCIETY
Secretary, Dr. Leo Harrington, 880 Ocean Ave., Brooklyn, N.Y. Meets monthly on fourth Tuesday, October to April.

BUFFALO RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph S. Gian-Francheschi, 610 Niagara St., Buffalo, N. Y. Meets second Monday of each month except during summer months.

CHICAGO ROENTGEN SOCIETY
Secretary, Dr. F. H. Squire, 1754 W. Congress St., Chicago 12, Ill. Meets second Thursday of each month October to April inclusive at the Palmer House.

CINCINNATI RADIOLOGICAL SOCIETY
Secretary, Dr. Samuel Brown, 707 Race St., Cincinnati, Ohio. Meets third Tuesday of each month, October to May, inclusive.

CLEVELAND RADIOLOGICAL SOCIETY
Secretary, Dr. D. D. Brannan, 11311 Shaker Blvd., Cleveland 4, Ohio. Meets at 6:30 P.M. at Allerton Hotel on fourth Monday each month, October to April, inclusive.

DALLAS-FORT WORTH ROENTGEN STUDY CLUB
Secretary, Dr. X. R. Hyde, Medical Arts Bldg., Fort Worth, Texas. Meetings held in Dallas on odd months and in Fort Worth on even months, on third Monday, at 7:30 P.M.

DENVER RADIOLOGICAL CLUB
Secretary, Dr. Edward J. Meister, 366 Metropolitan Bldg., Denver, Colo. Meets third Friday of each month at Denver Athletic Club.

DETROIT ROENTGEN RAY AND RADIUM SOCIETY
Secretary, Dr. E. R. Witwer, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne County Medical Society Building.

FLORIDA RADIOLOGICAL SOCIETY
Acting Secretary, Dr. Walter A. Weed, 204 Exchange Bldg., Orlando, Fla. Meetings in May and November.

GEORGIA RADIOLOGICAL SOCIETY
Secretary, Dr. James J. Clark, 478 Peachtree St., Atlanta, Ga. Meets in November and at annual meeting of Medical Association of Georgia in the spring.

RADIOLOGICAL SOCIETY OF KANSAS CITY
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month at a place designated by the president.

ILLINOIS RADIOLOGICAL SOCIETY
Secretary, Dr. Wm. DeHollander, St. John's Hospital, Springfield, Ill. Meets three times a year.

INDIANA ROENTGEN SOCIETY
Secretary, Dr. H. C. Ochsner, Methodist Hospital, Indianapolis. Meets annually second Sunday in May.

IOWA X-RAY CLUB
Secretary, Dr. Arthur W. Erskine, 326 Higley Bldg., Cedar Rapids, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. Special meetings by announcement.

KENTUCKY RADIOLOGICAL SOCIETY
Secretary, Dr. W. C. Martin, 321 W. Broadway, Louisville. Meets annually in Louisville on first Saturday in Apr.

LONG ISLAND RADIOLOGICAL SOCIETY
Secretary, Dr. Marcus Wiener, 1430-48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:30 P.M.

LOUISIANA RADIOLOGICAL SOCIETY
Secretary, Dr. J. R. Anderson, 1130 Louisiana Ave., Shreveport. Meets annually during Louisiana State Medical Society Meeting.

MICHIGAN ASSOCIATION OF ROENTGENOLOGISTS
Secretary, Dr. E. M. Shebesta, 1429 David Whitney Bldg., Detroit. Three meetings a year, Fall, Winter, Spring.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary, Dr. C. A. H. Fortier, 231 W. Wisconsin Ave., Milwaukee, Wis. Meets monthly on second Monday at University Club.

MINNESOTA RADIOLOGICAL SOCIETY
Secretary, Dr. Annette T. Stenstrom, 1218 Medical Arts Bldg., Minneapolis, Minn. One meeting a year at time of Minnesota State Medical Association.

NEBRASKA RADIOLOGICAL SOCIETY
Secretary, Dr. D. A. Dowell, Medical Arts Bldg., Omaha, Nebr. Meets third Wednesday of each month, at 6 P.M. at either Omaha or Lincoln.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. George Levene, Massachusetts Memorial Hospitals, Boston, Mass. Meets monthly on third Friday, Boston Medical Library.

RADIOLOGICAL SOCIETY OF NEW JERSEY
Secretary, Dr. H. R. Brindle, 501 Grand Ave., Asbury Pk. Meets annually at time and place of State Medical Society. Mid-year meetings at place chosen by president.

NEW YORK ROENTGEN SOCIETY
Secretary, Dr. Ramsay Spillman, 115 East 61st St., New York City. Meets monthly on third Monday, New York Academy of Medicine, at 8:30 P.M.

NORTH CAROLINA ROENTGEN RAY SOCIETY
Secretary, Dr. Major Fleming, Rocky Mount, N. C. An-

* Secretaries of Societies not here listed are requested to send the necessary information to the Editor.

nual meeting at time and place of State Medical Society. Mid-year scientific meeting at place designated.

North Dakota Radiological Society
Secretary, Dr. L. A. Nash, St. John's Hospital, Fargo. Meetings held by announcement.

Central New York Roentgen Ray Society
Secretary, Dr. C. F. Potter, 820 S. Crouse Ave., Syracuse. Three meetings a year. January, May, November.

Ohio Radiological Society
Secretary, Dr. Henry Snow, 1061 Reibold Bldg., Dayton, Ohio. Meets during annual meeting of Ohio State Medical Association.

Pacific Roentgen Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. L. E. Wurster, 416 Pine St., Williamsport.

Philadelphia Roentgen Ray Society
Secretary, Dr. R. P. Barden, University Hospital, Meetings first Thursday of each month from October to May inclusive at 8:15 P.M., in Thompson Hall, College of Physicians, 19 S. 22d St.

Pittsburgh Roentgen Society
Secretary, Dr. L. M. J. Freedman, 4800 Friendship Ave. Meets second Wednesday each month, 4:30 P.M., October to June, Pittsburgh Academy of Medicine.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Murray P. George, Strong Memorial Hospital. Meets monthly on third Monday from October to May, inclusive, 8 P.M. at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary Dr. A.M. Popma, 220 N. First St., Boise, Idaho.

St. Louis Society of Radiologists
Secretary, Dr. E. W. Spinzig, 2646 Potomac, St. Louis, Mo. Meets fourth Wednesday of each month, except June, July, August, and September, at a place designated by the president.

San Diego Roentgen Society
Secretary, Dr. Henry L. Jaffe, Naval Hospital, Balboa Park, San Diego, Calif. Meets monthly on first Wednesday at dinner.

San Francisco Radiological Society
Secretary, Dr. Martha Mottram, 450 Sutter St., San Francisco. Meets monthly on third Thursday at 7:45 P.M., first six months of year at Toland Hall, University of California Hospital, second six months at Lane Hall, Stanford University Hospital.

Shreveport Radiological Club
Secretary, Dr. R. W. Cooper, Charity Hospital, Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M., September to May inclusive.

South Carolina X-Ray Society
Secretary, Dr. T. A. Pitts, Baptist Hospital, Columbia, S. C. Meets in Charleston on first Thursday in November, also at the time and place of South Carolina State Medical Association.

Tennessee Radiological Society
Secretary, Dr. J. M. Frère, 707 Walnut St., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

Texas Radiological Society
Secretary, Dr. Asa E. Seeds, Baylor Hospital, Dallas, Texas. Next annual meeting, Temple, Texas, Jan. 17, 1945.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

University of Wisconsin Radiological Conference
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets every Thursday from 4:00–5:00 P.M., Room 301, Service Memorial Institute.

Virginia Radiological Society
Secretary, Dr. E. L. Flanagan, 116 E. Franklin St., Richmond, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. Thomas Carlile, 1115 Terry St., Seattle. Meets fourth Monday each month, October through May, College Club, Seattle.

X-ray Study Club of San Francisco
Secretary, Dr. J. M. Robinson, University of California Hospital. Meets monthly, third Thursday evening.

Cuba

Sociedad de Radiología y Fisioterapia de Cuba
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

British Empire

British Institute of Radiology Incorporated with the Röntgen Society
Medical Members' meeting held monthly on third Friday at 2:30 P.M. and Ordinary Meeting at same time on following Saturday, October to May, 32 Welbeck St., London, W.1.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets on the third Friday of each month at 4:45 P.M. at the Royal Society of Medicine 1, Wimpole St., London, W. 1.

Faculty of Radiologists
Secretary, Dr. M. H. Jupe, 32 Welbeck St., London, W. 1 England.

Section of Radiology and Medical Electricity, Australasian Medical Congress
Secretary, Dr. H. M. Cutler, 139 Macquarie St., Sydney, New South Wales.

Radiological Section of the Victorian Branch of the British Medical Association
Secretary, Dr. Keith Hallam, St. George's Hospital, K.E.W., Melbourne, E. 4, Victoria, Australia. Meets monthly from March to November inclusive.

Canadian Association of Radiologists
Secretary, Dr. A. D. Irvine, 540 Tegler Bldg., Edmonton, Alberta.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Radiological Section, New Zealand British Medical Association
Secretary, Dr. Colin Anderson, Invercargill, New Zealand. Meets annually.

South America

Sociedad Argentina de Radiologia
Secretary, Dr. Guido Gotta, Buenos Aires, Argentina. Meetings are held monthly.

Continental Europe

Sociedad Espanola de Radiologia y Electrologia
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7. Madrid, Spain. Meets monthly in Madrid.

Société Suisse de Radiologie (Schweizerische Röntgen-Gesellschaft)
Secretary for French language, Dr. A. Grosjean La Chaux de Fonds.
Secretary for German language, Dr. Scheurer, Molzgasse Biel. Meets annually in different cities.

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banul Mărăcine, 30, S. I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad: USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

Leningrad Roentgen Ray Society
Secretaries, Drs. S. G. Simonson and G. A. Gusterin. Meets monthly, first Monday at 8 o'clock, State Institute of Roentgenology and Radiology, Leningrad.

Moscow Roentgen Ray Society
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky. Meets monthly, first Monday, 8 P.M.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

THREE YEAR ROTATING SERVICE IN RADIOLOGY

The Peter Bent Brigham Hospital in Boston is announcing a three year rotating internship and residency in Radiology which began July 1, 1944. The service will include roentgenological training in Pediatrics and Obstetrics under Dr. E. B. D. Neuhauser at the Children's Hospital and Boston Lying-in Hospital, in pathology under Dr. Shields Warren at the Deaconess Hospital, and in Roentgen and Radium Therapy under Dr. Joseph H. Marks at the Deaconess Hospital. The initial six months and the final year of the combined service will be spent at the Peter Bent Brigham Hospital under Dr. Merrill C. Sosman. In individual cases where the candidate has had a thorough training in Pathology before starting this service, special arrangements can be made for study and original work in Radiation Physics, Nuclear Physics or Bio-Physics with radioactive tracers, either at Harvard University or Massachusetts Institute of Technology.

As soon as Procurement and Assignment will permit, two men will be accepted each year, one each on January 1 and July 1. Stipends of $500 the first year, $750 the second year and $1,000 the third year are paid, in addition to board, room, uniforms and laundry, or commutation for quarters, during each period. All of the hospitals are closely grouped around Harvard Medical School and all partake in the teaching of undergraduates and post-graduates in the School.

This combination of men and of hospital departments should do much to improve the basic training in Radiology and should help insure a continued output of well grounded young roentgenologists, for whom there is such a demand at present. The plans permit expansion to accommodate returning Service men who may desire a shorter period of training or review in parts of the field, as well as those who wish to complete the training which was interrupted by entry into the Services.

Vol. 53, No. 2

DEPARTMENT OF TECHNIQUE

Department Editor: Robert B. Taft, M.D., B.S., M.A., 103 Rutledge Ave.
Charleston, S. C.

A METHOD OF FOREIGN BODY LOCALIZATION USING TWO PARALLEL ROENTGEN FILMS

By SIEGFRIED W. WESTING, M.D.
Roentgenologist, Brooklyn Cancer Institute, Department of Hospitals,
NEW YORK, NEW YORK

THROUGH an article by Ashwin[1] I learned of a foreign body depth determination which impressed me as remarkably simple and new. On two parallel films make exposures from two different tube positions. On paper (see Fig. 2; solid lines) draw two horizontal lines, the distance of which equals the distance between the films. These lines are crossed by a vertical line. Starting at the vertical line enter on one of the horizontal lines the shift of the foreign body shadows, as visible on one of the films, and obtain point K. Repeat the procedure, using the other horizontal line and the other film, and obtain point K'. The extension of line KK' crosses the vertical line at N. The distance between N and the nearer horizontal line equals the distance between the foreign body and the film nearer the skin.

The determination of the depth of a foreign body—although always valuable and in certain cases sufficient—does not constitute a complete localization. In order to obtain complete localization one has to determine—in addition to the depth—the projection of the foreign body on the skin. Ashwin's procedure, notwithstanding the title of his article "A practical localisation method," requires supplementary steps before a localization method is obtained.

I have amplified Ashwin's procedure and thus developed a localization method which requires no special equipment, no arithmetical calculations, can be accomplished speedily and with minimal handling of the patient, and without roentgenoscopy. I understand that in hospital tents roentgenoscopy is not a simple matter during daylight. All that is necessary is two exposures on two parallel films and the drawing of a few straight lines on a sheet of paper.

The method calls for the following eleven steps:

(1) One film wrapped in a lightproof envelope is placed adjacent to the area to be examined. A second film in the same kind of envelope is held parallel to the first by a block of radiotransparent material (for instance, wood). Mark the second film "FAR," placing the letters as they appear in ordinary script, not mirror script.

(2) Measure the distance between the two films. This is the only measurement necessary. After this measurement has been performed once, it can be used for all subsequent localizations. This measurement has to be as accurate as possible.

(3) Adjust the roentgen tube as for an ordinary exposure. The anode film distance is optional.

(4) On the skin draw in ink a clock hand: the base of the clock hand at the point of incidence of the vertical ray; the point of the clock hand indicating the direction of the tube shift to be done later. Place a wire of identical shape and direction on the ink mark.

(5) Make the exposure. Shift the tube. (Amount of shift is optional. Direction of shift is optional as long as it is not straight toward or straight away from the foreign body. Any side shift will do. Direction has to conform to direction of ink-wire marker.) Make the second ex-

[1] Ashwin, C. Practical localisation method. *Radiography*, Lond., 1944, *10*, 6–7.

posure on the same two films. The patient has to keep motionless during the entire step (5).

(6) On paper (see Fig. 2) draw two parallel lines at a distance *exactly* equal to the distance between the films. Let these lines be crossed by a vertical line.

(7) After processing the films, hold the far film so that the word FAR will appear as ordinary script, not mirror script (see Fig. 1). Two shadows of the clock hand and two shadows of the foreign body will be seen. Of each shadow pair, consider first the shadow nearer the side to which the arrow points. Call the base of the first clock hand shadow *A*, of the second *B*; call one point of the first foreign body shadow *C*, and the identical point of the second foreign body shadow *D*. On the lower horizontal line, always starting at the vertical line, enter accurately *CD*, *AB* and *AC*. Thus the points *K*, *L*, and *M* will be obtained.

(8) Repeat step (7) using the near film with points *E*, *F*, *G*, *H* and upper horizontal line, thus obtaining the points *K'*, *L'*, *M'*.

(9) Draw lines *KK'* and *LL'* which cross the vertical line at *N* and *O*. Draw line *MM'* and the horizontal line through *N*; these lines cross at *P*.

(10) On the far film determine the direction of *AC*. If the wire marker and its shadow are assumed to be a clock hand pointing to 12 o'clock, the direction of *AC* can be described as the hour which would be indicated by the clock hand in this direction.

(11) The localization can now be reported: Place the part examined so that the skin carrying the ink drawing forms the upper surface Assuming that the clock hand points to 12 o'clock, proceed toward —— o'clock (direction *AC* determined on far film; 2:30 o'clock in this case) for ____ inches (equal to *NP*; $1\frac{9}{16}$ inches in this case). Thus the skin point vertically above the foreign body is arrived at. The foreign body is ____ inches (equal to *ON*; $2\frac{3}{8}$ inches in this case) vertically below this point. If operation is contemplated and the position during the intended operation is going to differ from the position just described, it may be advisable to repeat the localization in the position of the intended operation.

NEAR FILM FAR FILM

FIG. 1. Knee in maximal flexion. One metallic foreign body concealed in knee cavity. Factors: 68 kv., 10 ma., anode distance from near film 27 in., from far film 29 in. Tube shift 5 in. A 15° cone tilt was employed during the second exposure. Time: $2\frac{1}{2}$ seconds for each of the two exposures.

Ashwin makes the statement that to the best of his knowledge, his method has not previously appeared in *Radiography*. I have made a search of the literature and I have been unable to find the method published anywhere.

and have found it to yield a satisfactory degree of accuracy. At depths up to 6 inches an error of about 1/16 inch was observed; the range of error was even less with regard to the determination of the skin point vertically above the foreign body. Al-

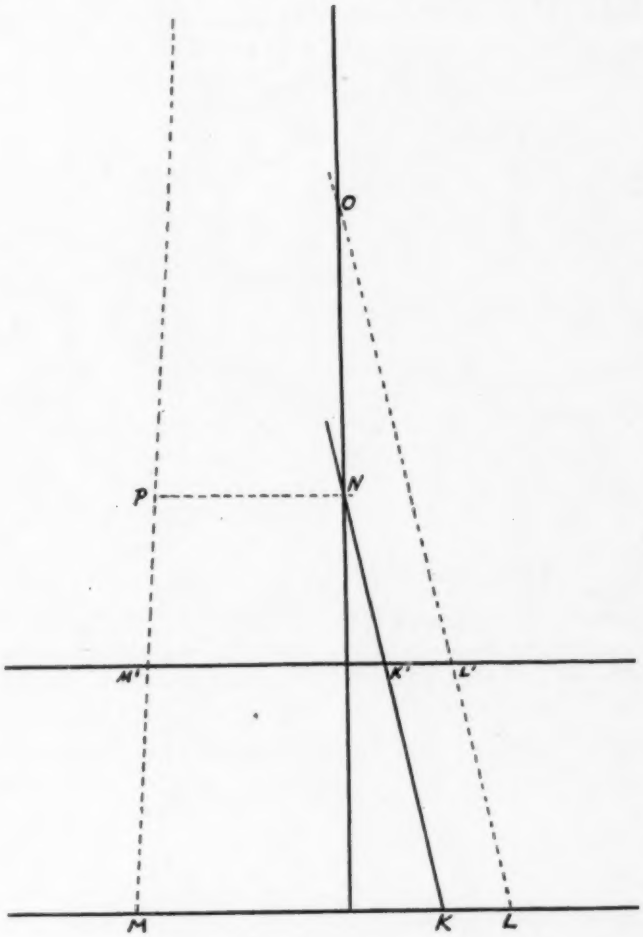


Fig. 2.

The method is applicable chiefly to not too heavy parts of the body. Should the method come into extensive use, manufacturers may undertake the production of suitable cassettes, plainly marked with the distance between the films and supplied with intensifying screens so that the method may also be employed for heavier parts.

The method presupposes that the upper surface of the body part under examination is horizontal or nearly so. I have used the method in the case of foreign bodies concealed in the knee cavity and on phantoms, though the method is very simple, it may be well to practice the method and check the results on a phantom before carrying out the actual localization.

ADDENDUM

Mathematical Proof of Ashwin's Depth Determination

I have worked out mathematical proof of Ashwin's principle and of my modified procedure and I am appending the proof for those interested in this phase of the problem.

Consider the situation which exists when two unselected exposures are made of a foreign body on two films, whose distance is a. The distance from the foreign body to the nearer film is f. One exposure creates foreign body shadows C' on the far film and G' on the near film, the other D' and H'. The lines $C'G'$ and $D'H'$ cross at the foreign body, I, and are located in one plane. This plane crosses the films in two parallel lines. One of these parallel lines contains C' and D', the other G' and H'. $C'D' \| G'H'$ (see Fig. 3). $G'H'/C'D' = IG'/IC'$.

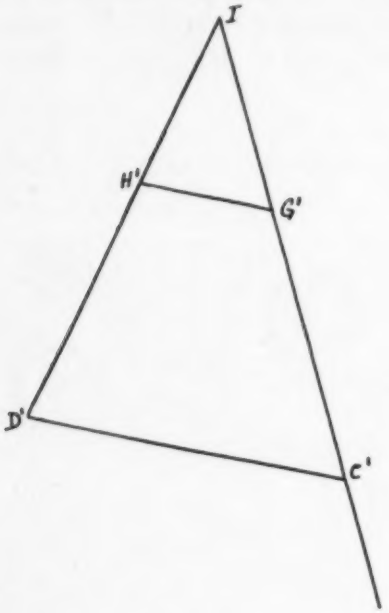


FIG. 3.

Next consider the plane which contains the line $IG'C'$ and the plumb line dropped from I. The plumb line crosses the films at $\mathcal{Q}$ and R. $I\mathcal{Q} = f$ and $\mathcal{Q}R = a$. This plane crosses the two parallel films in two parallel lines. One of these lines contains G' and $\mathcal{Q}$, the other C' and R. $G'\mathcal{Q} \| C'R$ (see Fig. 4). $IG'/IC' = I\mathcal{Q}/IR$ and, as shown above, $= G'H'/C'D'$.

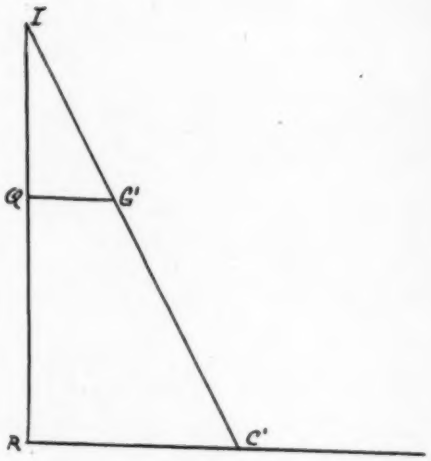


FIG. 4.

$C'D'$ and $G'H'$ are the shadow shifts. If different tube positions are used, a plane different from our first plane will result. In this plane the shadow shifts will have a length different from $C'D'$ and $G'H'$, but the relation between the two new shadow shifts will likewise be equal to $I\mathcal{Q}/IR$. No matter which two-tube positions are considered, the relation of the resulting shadow shifts will invariably be equal to $I\mathcal{Q}/IR$.

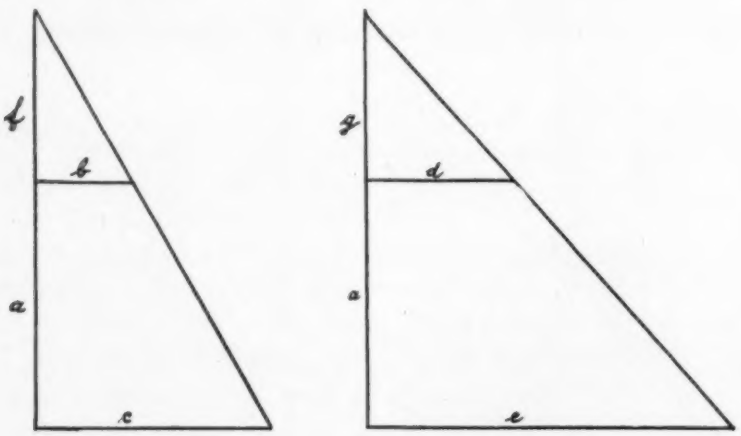


FIG. 5.

Let us call one pair of shadow shifts b and c, another one—produced by the same foreign body on the same films—d and e. $b/c=d/e$.

When each pair of shadow shifts is entered on a separate Ashwin line system (two horizontal, one vertical) (see Fig. 5), the following equations will result: $b/c=f/(f+a)$ and $d/e=g/(g+a)$. As shown above, $b/c=d/e$, therefore $f/(f+a)=g/(g+a)$. Therefore $f=g$. This means that entering on Ashwin's line system a pair of shadow shifts produced by one foreign body on one pair of films will always result in a line which has the length f.

The equation $f/(f+a)=b/c$ can be used to find f by calculation instead of drawing.

Mathematical Proof of the Present Modified Procedure

Consider the situation which exists during the first exposure as described in the first five directions. The vertical ray is a line which connects the anode, the skin marker, $\mathcal{J}$, its shadows E and A. $AE=a$, the distance between the films. The line which connects the foreign body shadows C and G passes through the foreign body, I, and crosses the first line at the anode. Both these lines are located in one plane, called hereafter "our plane." Our plane crosses the two films at right angles and in two horizontal lines. One of these lines contains A and C, the other E and G. $AC \| EG$. The vertical ray forms right angles with any line on the two parallel planes which passes through A or E. The vertical ray forms right angles with AC and EG. Since our plane crosses the skin at right angles, the vertical line from the foreign body to the skin is located in our plane. The intersection of this vertical and the skin is called S. Our plane crosses the upper skin sursurface in a horizontal line. This line contains $\mathcal{J}$ and S.

If one draws two parallel lines whose distance is a and lets them be crossed by a vertical line (see Fig. 6), he may consider these lines a reproduction of the vertical ray and of the crossing lines of our plane and the films. The crossing points of the vertical and the horizontal lines are reproductions of A and E. It is permissible to enter AC and EG on the horizontal lines. Point I must lie on the extension of CG and at distance f from EG, that means at the intersection of CG and a line running at distance f parallel to EG. This line crosses the vertical line at T. $\mathcal{J}$ is found through Ashwin's procedure whereby the shadow shifts of the skin marker are used. S is found as the intersection of a vertical extending from I and a horizontal running

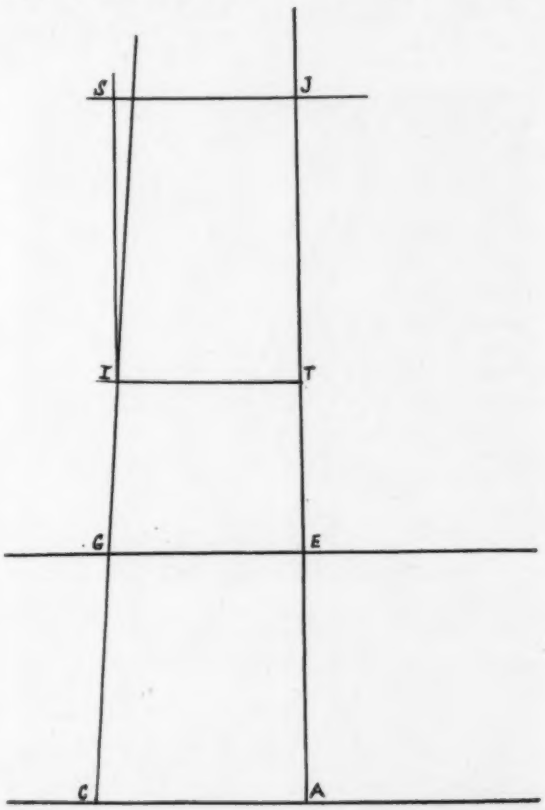


Fig. 6.

through $\mathcal{J}$ and representing the crossing line of the skin and our plane. $S\mathcal{J}TI$ is a rectangle. $S\mathcal{J}=TI$. Since Figure 6 turns out to be a true replica of Figure 2, it is proved that NP is equal to the distance between the skin marker $\mathcal{J}$ and the projection of the foreign body on the skin S. Since $\mathcal{J}S$ is parallel to AC, the direction of $\mathcal{J}S$ on the skin is identical with the direction of AC on the far film.

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A DEVICE FOR COMPRESSION POLYGRAPH STUDIES OF THE GASTROINTESTINAL TRACT

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TO RECORD roentgenoscopic observations instantaneously, Berg introduced a spot film compression device into the roentgenography of the gastrointestinal tract. However, most of the roentgen apparatus does not lend itself easily to the installation of this equipment, which, in addition, is quite expensive. A substitute for the spot film compression device consisting of compression polygraphs has proved very satisfactory, and a home made apparatus was constructed for this purpose.

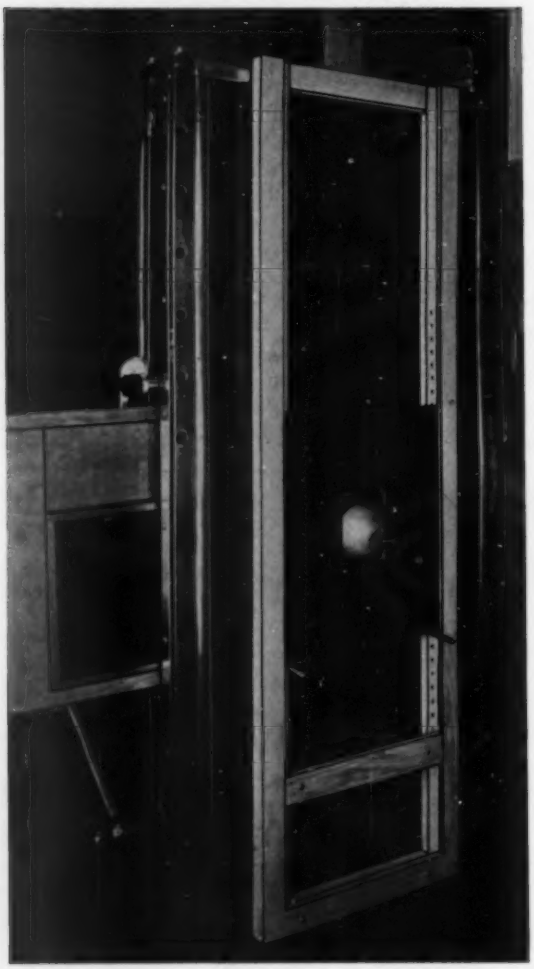

FIG. 1. The hanging frame, cut-out lead shield with compression cup in the erect position ready for use.

A home made Bucky polygraph, which was described by Nathanson and Wittenberg,[2] was further developed so it could be utilized to obtain compression polygraph studies in the erect and prone positions.

The cut-out lead shield and wooden frame for the Bucky polygraph were made according to the specifications in the previously mentioned article. An aluminum cup which fits snugly into the opening of the lead shield was added for compression purposes. In order to use the lead shield in the erect position, two wooden bars which are connected by crossbars were constructed. These bars contain a channel ½ inch deep in which the lead shield moves freely. Openings are provided within the bars 1 inch apart. The lead shield is inserted from the top, and at the desired level it is held in place by two wooden pegs or screws which fit into the openings. The length of the entire frame is 5 feet. Angle irons are attached to the top of the wooden frame which make it possible to lift the frame off the table easily.

To do an erect compression polygraph, the wooden frame is placed over the table, the lead shield is inserted from the top and secured at the desired level by the wooden pegs, and the compression cup is placed into the cut-out lead shield. The patient is then roentgenoscoped and the lead shield adjusted to the correct level under roentgenoscopic control. Compression polygraphs can now be taken. The patient is instructed to rest against the cup, to use

slight pressure, to press hard, and very hard. Thus four roentgenograms are obtained with a varied degree of compression. in the erect position, but a varied amount is also obtained by instructing the patient to use a varied amount of pressure. The

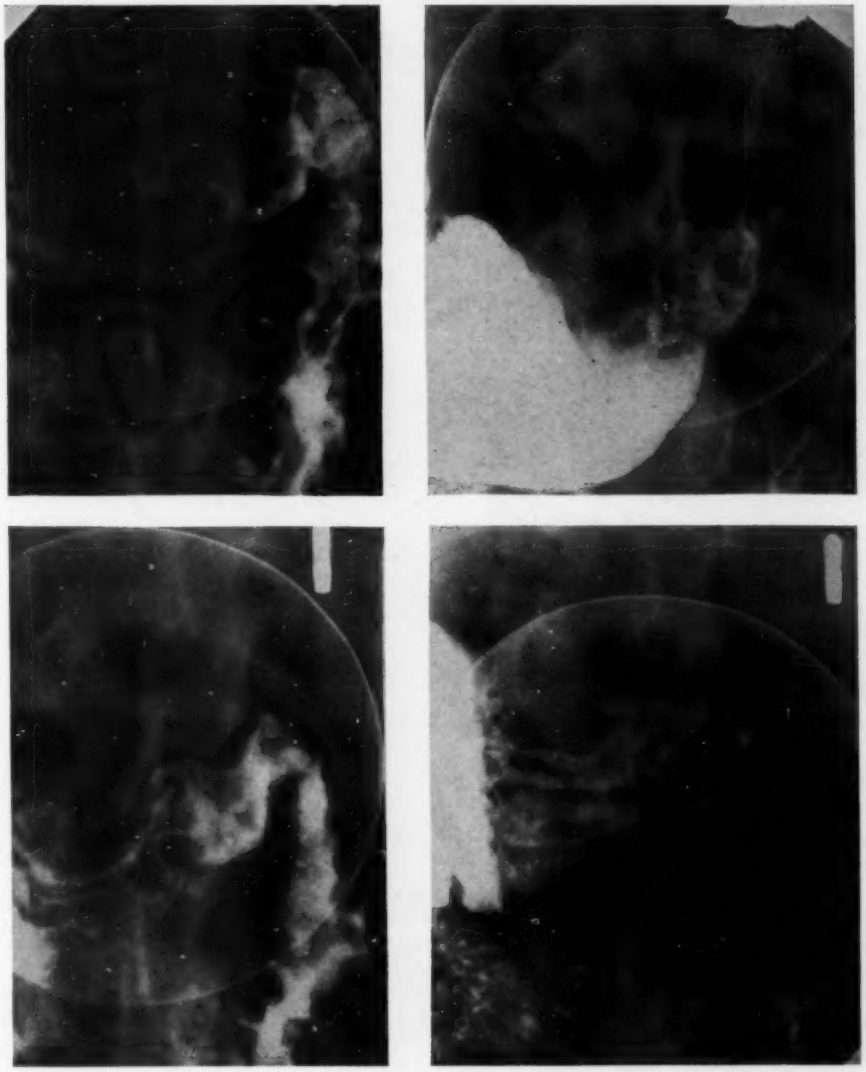

Fig. 2. Prone compression polygraph of the duodenal bulb in a case showing a residual ulcer crater with convergence of the mucosal folds in an undeformed duodenal bulb.

In the prone position the wooden frame is unnecessary. The cut-out lead shield and compression cup are only needed to obtain the compression studies. However, compression cannot be regulated as easily as roentgenograms obtained by this method have been very satisfactory, and it has been very gratifying to demonstrate pathologic conditions which otherwise would have been hidden. The goal of Berg[1] and Åker-

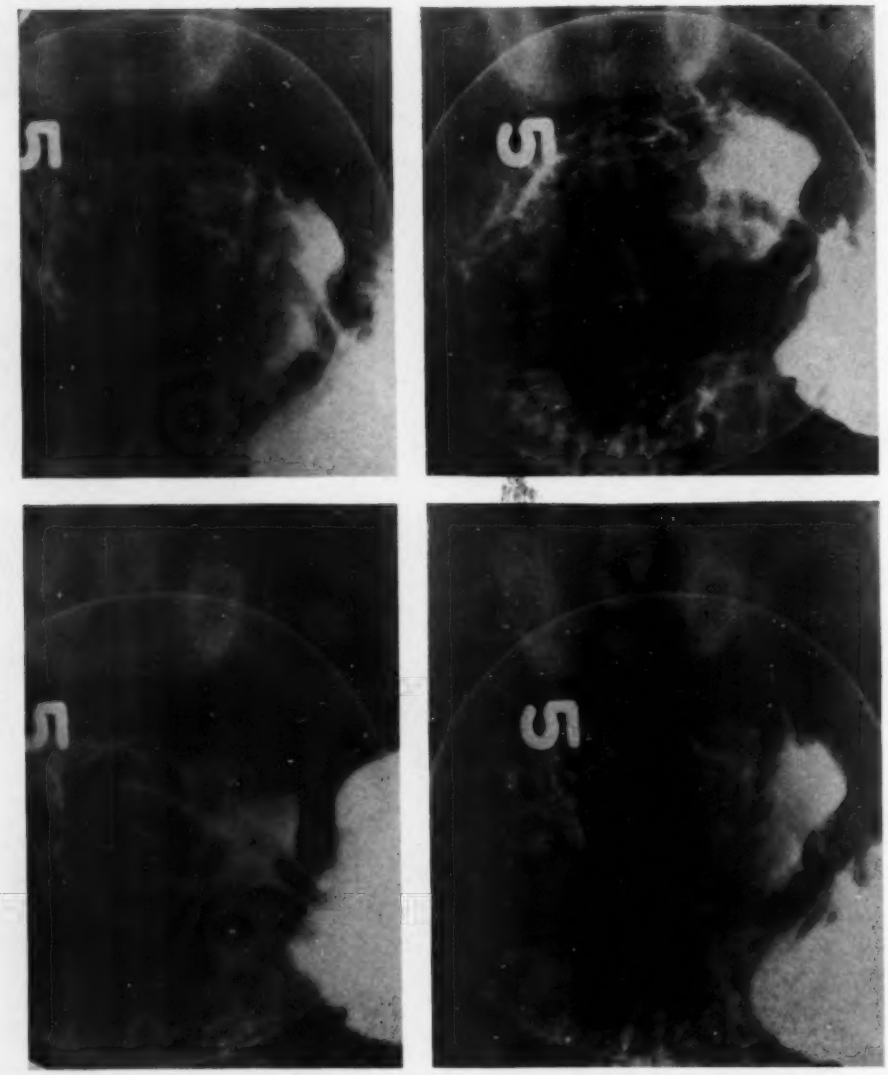

FIG. 3. Erect compression polygraph of the duodenal bulb in a case showing a minimal marginal deformity and distortion of the mucosal pattern. These cases illustrate the various degrees of compression which can be obtained with the device.

lund to use measured compression for mucosal studies of the gastrointestinal tract is accomplished by the aid of a very simple apparatus which represents a material cost of approximately five dollars, and can be attached to any roentgenographic table which has a Bucky tray.

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REFERENCES

1. BERG, H. H. Röntgenuntersuchungen am Innenrelief des Verdauungskanals. Georg Thieme, Leipzig, 1930.
2. NATHANSON, L., and WITTENBERG, M. M. Home made Bucky polygraph. AM. J. ROENTGENOL. & RAD. THERAPY, 1942, *47*, 946–947.

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ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

ROENTGEN DIAGNOSIS

HEAD

Hartley, J. Blair, and Burnett, C. W. F. The radiological diagnosis of craniolacunia. *Brit. J. Radiol.*, April, 1943, *16*, 99–108.

Craniolacunia is a developmental anomaly of the bones of the vault of the fetal skull. The bones at the base are normal. There are depressions on the inner surface of the bones of the vault, and in a more pronounced form, called craniofenestria, there are actual openings in the bones through which brain substance sometimes protrudes. This condition is usually associated with other defects, such as spina bifida, encephalocele, hydrocephalus or talipes.

If there is hydramnios or hydrocephalus during pregnancy an antenatal roentgen examination should be made for this condition. At birth its existence should be suspected in the presence of any of the above-named anomalies.

The author reviews 28 cases seen in a year's investigation, showing that the anomaly is much more frequent than has been believed. The lacunae tend to disappear as the child grows and the anomaly in itself does not constitute a danger to life. There is some evidence that it is familial or hereditary and it would be interesting to follow up these cases which might contribute valuable knowledge to the study of heredity.—*Audrey G. Morgan.*

NECK AND CHEST

Lindsay, John R., Templeton, Frederic E., and Rothman, Stephen. Lesions of the esophagus in generalized progressive scleroderma. *J. Am. M. Ass.*, Nov. 20, 1943, *123*, 745–750.

Generalized scleroderma is a systemic disease of the connective tissue. In most cases the process starts on the hands and feet. Three stages can be distinguished clinically: the edematous, the indurative and the atrophic stage. The initial edema does not pit on pressure. The skin appears tense and cannot be folded. In the second phase the skin hardens and stiffens. In the third stage the tips of the fingers become smaller and pointed, and the fingers are immobilized in a flexed position. The face, the nose, the ears, and the lips become gradually smaller and thinner. All mucous membranes of the mouth may be involved in the indurative and atrophic process. Ulcers, probably due to tension and deficient circulation, develop mainly on the finger tips, the elbows and the ankles. Fibrosis of the lungs without any subjective symptoms is a common sign of the disease. Two types of the disease can be distinguished: the type starting with Raynaud-like signs and symptoms and the arthritic type, the latter starting with joint pains and stiffness. These two types are often intermingled.

Pathologically, the main feature of the disease is a peculiar change of the collagenous tissue usually designated as homogenization. After the edematous stage, with the picture of swollen and loose fibers, has subsided, the collagenous fibrillae become thickened and less acidophilic than normal. In this dense fibrous connective tissue the fibroblasts are shrunken and less numerous than normal. The vessels are intensely involved by a fibrotic process. The muscularis is completely replaced by connective tissue consisting of gross fibers.

Lesions of the Esophagus in Generalized Scleroderma. A review of the literature indicates that difficulty in swallowing has been reported in 16 cases of diffuse scleroderma. The authors report 5 cases which were clinically and histologically typical cases of diffuse scleroderma. They presented clinical evidence of a disturbance of the swallowing mechanism, varying apparently only in the stage of the esophageal lesion. A frequent complaint was of burning pain behind the sternum about an hour after meals, worse on lying down, probably due to regurgitation of gastric contents into the esophagus and resulting chronic esophagitis. Other esophageal symptoms consist of inability to swallow more than a few mouthfuls of fluid rapidly, difficulty in swallowing while lying down and a sensation of fullness behind the sternum or of a weight on the chest for a few moments after swallowing. These symptoms are explained by the roentgenoscopic observation that normal peristalsis is absent in the lower two-thirds of the esophagus and that the

contents accumulate in this relaxed and dilated portion. Propulsion of the bolus occurs mainly by gravity or by the transmitted effect of peristalsis in the upper end on repeated swallowing.

On roentgenoscopy 3 of the patients were found to have a localized narrowing of the esophagus about 4 or 5 cm. above the level of the diaphragm. In all 5 patients there were pronounced changes in the muscular movements. With the patient standing, barium sulfate passed from the pharynx to the stomach with some delay at the site of stricture but not at the cardia. When the patient was placed in the horizontal position the barium sulfate entered the esophagus and there remained. As each successive bolus was taken, the esophagus distended to receive it, reaching a width of approximately 4 to 5 cm. The primary peristaltic wave which normally arises in the pharynx and travels the length of the esophagus with each act of deglutition traveled only down to about the level of the suprasternal notch. If these patients were allowed to remain in the horizontal position after the esophagus filled and additional barium sulfate was given, barium sulfate entered the stomach. The amount of barium sulfate that entered the stomach with each act of deglutition seemed to be equivalent to the amount taken with each swallow.

Diffuse chronic esophagitis extending above the level of the aortic arch was a constant finding on esophagoscopy. This might be due to an inflammatory change in the mucosa comparable to that seen in the skin occurring on the basis of scleroderma or to a peptic esophagitis secondary to regurgitation and retention of gastric contents. Another esophagoscopic finding was that of chronic ulceration in the lower third of the esophagus down to the phrenic ampulla and stenosis at the upper limit of the ampulla. Roentgenoscopy indicated that the narrowing of the esophagus above the phrenic ampulla is constant. It is seen during expiration as well as during inspiration. The chronic ulceration was probably a direct result of the esophagitis, with sclerodermatic changes as a predisposing factor. The tendency toward stricture in these cases appears to be explained as a direct result of the chronic ulceration. The limitation of the stricture to the area of greatest ulceration just above the ampulla and the tendency of chronic ulceration in the esophagus at any level to produce stenosis support this view.—*S. G. Henderson.*

Williams, E. Rohan. Dilatation of oesophagus. *Brit. J. Radiol.*, July, 1943, *16*, 220.

The greatest degrees of dilatation of the esophagus are seen in cardiospasm. Sometimes in old cases the esophagus is not only dilated but elongated and becomes tortuous and kinked. Roentgenograms of 4 such cases are given and details of the roentgen appearances discussed.

The appearance of the dilated esophagus without contrast medium depends on its fluid content. In 1 of the above cases with a fluid content the esophagus is relatively opaque while in another almost entirely occupied by air the hypertrophied wall of the esophagus is clearly seen against the lung field. A clean-cut fluid level is seldom seen in plain roentgenograms, probably because of the collection of froth and lighter food materials at the top of the fluid column.—*Audrey G. Morgan.*

McLaren, John W. Kymography and its application to oesophageal movement. *Brit. J. Radiol.*, Sept., 1943, *16*, 270–273.

It has been commonly supposed that food moves down the esophagus by peristalsis, that is, a coördinated reaction in which a wave of contraction preceded by a wave of relaxation passes downward, the food always entering a segment which has relaxed actively and enlarged to receive it.

The difficulties of screen observation and cinematography in studying the movements of the esophagus are pointed out and an analysis given of the movements of the esophagus by kymography with a fixed grid and moving film. Kymograms are reproduced and the method of transforming them to graphs showing the movements of the esophagus illustrated. These graphs show that a band of contraction passes down the esophagus at a speed that varies somewhat in different individuals. The average is about 3 to 3½ inches in two seconds. As the band contracts it may increase in length and appear to push the food along, but there is no band of relaxation preceding it. While negative pressure was not demonstrated, it is not inconsistent with the findings. There was no positive evidence of an eccentric wave of contraction. In one case in which there was a suggestion of it the kymograph was not good enough to be analyzed by this method. The findings show that the movement of the esophagus is of a different type from the classical peristalsis. None of the kymograms were good enough to permit

of analysis of the movements of the diaphragmatic opening. A further study should be made of the movements shown in pathological conditions.—*Audrey G. Morgan.*

TRIMBY, ROBERT H. Congenital laryngeal stridor; lack of relation to thymic diseases. *J. Am. M. Ass.*, July 10, 1943, *122*, 740–742.

There exists a widespread tendency to place the blame for a respiratory abnormality of any magnitude in infancy on the thymus gland, particularly if there is roentgen evidence of thymic enlargement. Congenital laryngeal stridor, the most frequently encountered form of chronic stridor in infancy, has been recognized as an entity for many years. Although it is not often encountered, it is worthy of attention, since it it apt to cause anxiety owing to its being mistaken for some more serious disease.

The larynx is fully developed at birth and increases in size to puberty, with no sex difference noted in infancy. The structures about the upper aperture are soft and readily collapsible. The epiglottis, cartilages of Wrisberg and apexes of the arytenoid cartilage are composed of yielding, elastic cartilage. The rest of the larynx is of brittle hyaline cartilage. With the advent of direct laryngoscopy, the pathologic physiology of congenital laryngeal stridor was directly observed. McKenzie in 1925 reported direct laryngoscopic observations made by D. R. Paterson in which the latter saw "with each inspiration the soft parts of the cricoid, including the arytenoids and interarytenoid folds, 'disappear,' to so speak, into the interior of the larynx, being drawn downward and forward and to such an extent in pronounced cases that the upper border of the cricoid could be seen as a transverse ridge through the stretched mucous membrane." The sides of the orifice were also observed to be sucked in, and when he held one side up with forceps the noise stopped at once. Hill in 1930 described the situation as an exaggeration of the infantile type of larynx, differing from normal only in degree, and he believed that the stridor was probably due to the vibrations of the flabby epiglottis and unsupported arytenoepiglottic folds being sucked inward on inspiration. The pathogenesis described by McKenzie and by Hill seems fulfilled in cases cited by the author.

Although congenital laryngeal stridor is the most common cause of stridor in early infancy, it must be differentiated from other congenital abnormalities which interfere with respiration, such as a small glottic lumen, congenital web below the glottis, micrognathia, macroglossia, laryngeal papilloma and cysts. Laryngeal spasm, perilaryngeal abscess, mediastinal tumor, foreign body and at times acute infections are important considerations. Simple thymic enlargement is rarely responsible for respiratory embarrassment in infancy.

Since congenital laryngeal stridor is usually outgrown, active therapy is seldom required.—*S. G. Henderson.*

KLEIN, NORBERT. Cervical sympathico-blastoma. *Brit. J. Radiol.*, June, 1943, *16*, 183–184.

Malignant tumors originating from the cells of the sympathetic nervous system are generally found in the adrenals or in the retroperitoneal lymphatic glands. This article describes and illustrates a case in a boy of sixteen in which the primary tumor was on the right side of the neck near the angle of the jaw. Such cases of sympathicoblastomas originating from the cervical sympathetics are rare. There were numerous metastasesin the bones of the shoulder girdle and in the skull. The former had resulted in a pathological fracture of the surgical neck of the left humerus which was the lesion that brought the patient for examination.—*Audrey G. Morgan.*

CAMPBELL, THOMAS A., STRONG, PAUL S., GRIER, GEORGE S., III, and LUTZ, RAYMOND J. Primary atypical pneumonia. *J. Am. M. Ass.*, July 10, 1943, *122*, 723–729.

The purpose in this report is to emphasize the epidemiologic importance of primary atypical pneumonia, to consider its clinical features with special reference to the roentgenologic findings and to attempt to clarify the involved pathologic processes, omitting entirely the problem of etiology.

Epidemiology. It seems certain that the disease tends to occur both endemically and in minor epidemics. While the increased frequency with which roentgenograms have been taken recently has undoubtedly accounted for the recognition of many cases, there has been an actual increase in the incidence of the disease at the Army post from which this report comes as well as in neighboring civilian hospitals. The number of cases at this post increased from 17 in March to 105 in October. Because of the long

period of hospitalization this disease gives rise to a great loss of man-days and it therefore becomes of much military importance when it reaches epidemic proportions. Very few data were obtained on the incubation period of the disease, but most investigators estimate this period to be about seven to twenty-one days.

Clinical Observations. A gradual onset was noted in the majority of patients and was associated with prodromal symptoms of non-productive cough, low grade fever, muscular aching, malaise, headache and an occasional sore throat. The most common early symptom was a dry cough, which later became productive and often paroxysmal. Substernal soreness on coughing was a common and often distressing symptom. The most significant and frequently the only abnormal initial chest signs were suppressed breath sounds. The majority of the white blood cell counts were normal. A definite leukopenia was rare. No predominating pathogenic organisms were found in the sputum except in 3 instances, and these were all pneumococci of higher types which were considered to be without causative significance. All blood cultures were sterile. The temperatures on admission ranged from normal to 105° F., averaging 102° F., and fluctuated widely over twenty-four hour periods. The maximum fever was frequently reached within thirty-six hours after admission and terminated by lysis in about five days, regardless of the form of therapy. A few patients continued to have fever for as long as twelve to eighteen days. Spread of the disease process from one lobe to another or from one lung to the other was noted occasionally. The average period of hospitalization was twelve days, partially due to the necessity of keeping these patients in the hospital until they are ready to assume full military duty.

Sulfathiazole and sulfanilamide did not alter the course of the disease in any way. Routine therapy was supportive in nature and included bed rest, adequate fluids, antipyretics and sedatives.

Roentgenologic Aspects. An analysis of the roentgenograms demonstrated that 81 per cent of the pulmonary lesions were basilar, while 11 per cent of the cases showed involvement of the right upper lobe and in 5 instances were misdiagnosed as pulmonary tuberculosis. In 100 of these cases in whom adequate progress roentgenographic studies were made it was found that 57 per cent had cleared roentgenographically at the end of ten days, 85 per cent in twenty days and the remaining 15 per cent in the ensuing twenty day period.

Careful examination of the basilar shadows revealed a streaking type of density which radiated downward from the lung root and extended outward over the leaf of the diaphragm conforming to the general distribution of the bronchi. Superimposed on the stringy density was a mottled type of shadow which occurred in any part of the chest but was most commonly seen at the right cardiophrenic angle at or below the lung roots or along the left border of the heart. The mottled densities were interpreted as a lobular form of atelectasis based on a definite mechanical factor—the complete block of the smaller bronchi and bronchioles by collected exudate and swollen epithelium. It was thought that the stringy type of densities represented a combination of exudation within bronchi and bronchioles, swollen bronchial and bronchiolar epithelial lining, and peribronchial and peribronchiolar cellular infiltration. On rare occasions a complete lobe became atelectatic, and in these roentgenograms the shift of the mediastinum and the elevation of the diaphragm were pronounced. The collapsed lobe often simulated lobar pneumonia, but the diminished volume of the involved lobe, narrowing of the rib interspaces, shift of the mediastinum and elevation of the diaphragm revealed its true atelectatic nature. In many cases the mottled densities were seen to appear and disappear over night. The intermittent obstruction of the smaller bronchi and bronchioles was thought to be responsible for the transient pulmonary changes. One striking feature that was quite apparent in the majority of cases was the presence of horizontal linear densities overlying either diaphragm, usually after considerable clearing of the basilar shadows. These band densities had the appearance of the platelike atelectasis described by Fleischer and his associates.—*S. G. Henderson.*

Simon, G. Some problems in connection with the early x-ray changes in adult pulmonary tuberculosis. *Brit. J. Radiol.*, July, 1943, *16*, 217–219.

The author discusses a group of cases of adult tuberculosis in which there were no clinical signs and only slight roentgen signs. The lesions were of two types: (1) Round foci about 1 cm. in diameter or small smudges of

opacity, not so well defined. Both of these appearances were seen in the subclavicular region. (2) Mottled opacities of low density about 1–3 mm. in diameter below the clavicles or in the mid-zone. Such slight signs are often considered of no importance if there are no clinical signs but the author's material shows that while the round focus is comparatively benign if there are no clinical symptoms, a third of the smudge foci will progress to clinical tuberculosis and rather more than that of the mottled foci. In the cases that progress, present methods of treatment are inadequate and it is very important that these cases be brought to the attention of the clinician.

A more complete study should be made of the prognosis in the cases with only slight changes. A large series of cases should be reported by different authorities. Possibly a central registry similar to the American Bone Tumor Registry would be a good thing. More of these slight lesions than before have appeared since the introduction of mass roentgenography.—*Audrey G. Morgan.*

WARFIELD, CHESTER H. Types of pulmonary tuberculosis which demand disqualification for active duty in the Navy. *Radiology*, Sept., 1943, *41*, 282–283.

The object of this article is to acquaint civilian radiologists with the problems presented to radiologists in the Navy by the Bureau of Medicine and Surgery of the Navy Department and to enable them to explain to family and friends the reasons why some men without manifest lesions are returned home as unfit for service in the Navy.

All men, including officers, who present themselves for service in the Navy have a roentgen examination of the chest made. A 35 mm. film and the photofluoroscopic method are first used. This merely shows the presence of a pathological lesion, if any, and such lesions are overlooked in less than 1 per cent of the cases. If there is any pathological condition a 14×17 inch film is taken. If the lesion seen on the 35 mm. is confirmed the man is sent to the Naval Hospital for study and a decision as to what shall be done with him.

In April, 1941, the Surgeon General of the Navy outlined the following roentgen findings as sufficient cause for disqualification for service in the Navy: (1) any evidence of reinfection (adult) type tuberculosis, active or inactive, exclusive of slight thickening of the apical pleura; (2) evidence of active primary (childhood) tuberculosis; (3) inactive primary pulmonary tuberculosis if the degree or extent of involvement appears to be of present or future clinical significance; (4) extensive multiple calcification in the lung parenchyma and massive calcification at the hilum; (5) evidence of fibrinous or serofibrinous pleuritis.—*Audrey G. Morgan.*

BRAILSFORD, JAMES F. The radiographic postero-medial border of the lung, or the linear thoracic paraspinal shadow. *Radiology*, July, 1943, *41*, 34–37.

The author discusses work done on the linear thoracic paraspinal shadow at the University of Birmingham, Birmingham, England. This line is frequently seen on anteroposterior roentgenograms of the dorsolumbar area on the left side and less frequently on the right side. It extends upwards sometimes to the level of the fourth dorsal vertebra, but only as far as the reflection of the pleura on to the diaphragm, for it marks the postero-medial border of the lung and is caused by the sharp contrast between the relative radiotransparency of the lung and the opacity of the vertebral tissues to the rays. A diagram is given showing a transverse section of the body at the level of the ninth dorsal vertebra and the relationship of the postero-medial lung surface to the vertebrae. Tracings are also given from roentgenograms showing the postero-medial border of the lung in various conditions, including paravertebral abscess, hematoma associated with crush fracture of the eleventh dorsal body, osteochondritis of the dorsal spine and an abscess associated with collapse of the ninth vertebral body. In Paget's disease localized to one or more dorsal vertebral bodies the postero-medial border of the lung is deviated laterally by expansion of the bodies of the vertebrae. Any inflammatory or neoplastic change in the bodies of the vertebrae or the areolar tissue surrounding them may cause such deviations and it may be necessary to use other methods of examination to determine the cause of the deviation.—*Audrey G. Morgan.*

GARLAND, L. HENRY. The postero-mesial pleural line. *Radiology*, July, 1943, *41*, 29–33.

Several months ago an editorial appeared in *Radiology* which discussed the correct interpretation of a slender marginal line of demarcation which can sometimes be seen on sagittal roentgenograms of the lower posterior thorax, es-

pecially on the left side, though it may also appear on the right side. This editorial suggested that the line might be caused by an anomalous vessel.

Roentgen-anatomical studies by Lachman and a review of some cross-section anatomical texts have convinced the author that this line is not caused by an azygos vein but by a projection of the postero-mesial border of the lung and pleura and that it may therefore properly be called the postero-mesial pleural line.

Study of a number of cross-section anatomical texts showed that the border of the lung did lie in the sagittal plane at some level between the fifth and twelfth thoracic segments in about one-fourth of the cases.—*Audrey G. Morgan.*

Sosman, Merrill C. The x-ray in curable heart disease. *Radiology*, Oct., 1943, *41*, 351–362.

Heart disease is generally considered incurable but it is of interest and value to physician, radiologist and patient to know that there are certain forms of heart disease that are curable by medicine or surgery. Three forms that are curable by surgery are heart disease due to hyperthyroidism, patency of the ductus arteriosus and chronic constrictive pericarditis. Three that can be relieved by medicine are beriberi heart disease, heart disease in acute nephritis and anemia, and in gummatous myocarditis. Cases of these different forms of heart disease are described and illustrated with roentgenograms. The clinical and roentgen signs are reviewed.

Heart disease due to hyperthyroidism shows marked hypoactivity on roentgenoscopy. The heart is usually considerably enlarged, rather triangular in shape like a medium-sized pericardial effusion, while the heart beat is indistinct and of small amplitude. These signs are overcome by treatment with thyroid extract. In patent ductus arteriosus the heart is usually enlarged, especially the left ventricle, the pulmonic artery is more prominent than usual, projecting anteriorly and to the patient's left, the hilar vessels are enlarged, especially on the right side, in less than half the cases pulsation of the hilar vessels can be seen roentgenoscopically, the so-called "hilar dance"; the left auricle was dilated posteriorly in nearly all of the author's cases; there is striking hyperactivity of the left ventricle and an associated increased expansile pulsation of the dilated pulmonary artery, synchronous with the hyperactive ventricular systole. These signs decrease after ligation of the ductus.

In constrictive pericarditis plaques of calcification can be demonstrated on roentgenoscopy; occasionally they dance with systole like a calcified heart valve. By rotating the patient it can be shown that the calcification is on the surface of the heart and not in its interior. The commonest places for calcification to occur are in the auriculoventricular sulcus, where it may form a complete ring around the heart, and on the diaphragmatic surface of the pericardium. Differential diagnosis from other similar conditions can be made by careful roentgen examination, particularly roentgenoscopy. In beriberi heart disease roentgen examination may show a hyperactive heart beat. If there is much edema there may be pericardial fluid which masks the heart beat. The amplitude of the beat on the right and left sides should be compared carefully in both posteroanterior and oblique positions. If pericardial effusion is suspected, it is important to compare the pulsation of the left ventricle with that of the aorta. The hyperactivity of the left ventricle may be masked by effusion but the expansion and collapse of the aorta may be definitely increased in amplitude.

The enlargement of the heart seen in acute nephritis may be due to hydropericardium or acute dilatation or both, while that in anemia is due to dilatation. In the latter case the heart will show hyperactivity of the myocardial beat. With treatment of the primary disease the heart will return to normal size, shape and activity. Heart failure due to pernicious anemia is also reversible. In gummatous myocarditis the roentgen changes are striking and the improvement after antisyphilitic treatment most marked.

Careful attention to detail in roentgen examination should be given in every case of heart failure in order to determine those cases in which successful treatment is possible.—*Audrey G. Morgan.*

ABDOMEN

Weinberg, Tobias B., and Raider, Louis. Pedunculated tumors of the stomach prolapsing through the pylorus. *Radiology*, July, 1943, *41*, 52–55.

Pedunculated tumors of the stomach are of four types: solitary benign tumor, pedunculated carcinoma, part of a multiple polyposis and

prolapsed mucosa. These prolapsing tumors constitute a fraction of 1 per cent of all tumors of the stomach. They may prolapse through the pylorus into the duodenum.

There is no typical clinical picture. A characteristic roentgen finding is a filling defect, the location of which depends on the site of the polyp at the time of examination. Delayed emptying often results and there may be a considerable six hour residue. When the tumor is in the duodenum the pyloric end of the stomach appears normal.

A case is described in a man of seventy-five. When the first roentgen examination was made on April 3, 1941, the stomach was normal in size and position with no evidence of intrinsic disease. There appeared to be extrinsic pressure along the lesser curvature. There was a persistent rounded filling defect in the bulb of the duodenum. A diagnosis was made of polyp in the duodenum and extrinsic pressure on the stomach by the liver. The patient left the hospital but was readmitted November 7, 1941, with complaints similar to those on his first admission. But he had grown worse and had lost 15 pounds since his discharge. The roentgenogram now showed an irregular defect in the lesser curvature at the pyloric end of the stomach. A diagnosis was made of a mobile pedunculated polyp of the pylorus capable of prolapsing into the duodenum. The patient died on February 8, 1942, and autopsy showed a benign polyp of the pyloric region of the stomach. The liver was enlarged and full of nodules which were found on microscopic examination to be multiple primary carcinomata of the intrahepatic bile ducts.—*Audrey G. Morgan.*

ROENTGEN AND RADIUM THERAPY

WASSON, W. WALTER. Intravaginal roentgen irradiation of cancer of the cervix. *Radiology*, May, 1943, *40*, 454–457.

A new apparatus for intravaginal roentgen treatment of cancer of the cervix is described and illustrated. Cylinders made of brass are used to limit the distribution of the rays. The cylinders vary from 2 to 4 cm. in diameter and one is selected that fits the vagina to be treated. A proctoscopic light is fitted to the cylinder and the vagina inflated to place the cylinder properly. This apparatus can also be used in routine examination of the vagina.

The apparatus that contains the roentgen tube is brought into contact with the cylinder and clamped in position. A periscope is used for checking the position of the cylinder after the apparatus and the table are clamped in position. The procedure takes little more time than is required for the insertion of a vaginal speculum and causes the patient no discomfort. Any desired kilovoltage and any length of exposure can be used. The author has found that 200 kv. with the usual factors is the preferred voltage.

A diagrammatic sketch is given showing the relationship of the cylinders to the area treated and the uterine adnexa. This method makes cervical cancer a superficial lesion and makes uniform distribution of the irradiation in the female pelvis possible.—*Audrey G. Morgan.*

SCHMITZ, HERBERT E. Further study of supervoltage x-ray therapy in carcinoma of the cervix. *Radiology*, May, 1943, *40*, 458–462.

It has been ten years since the first treatment of cancer of the cervix was given with 800 kv. maximum irradiation. The results of this treatment since that time are discussed and 2 cases reported in which unusual metastases occurred. The first case was in a woman of thirty-two who was treated by the supervoltage method for a carcinoma of the cervix of Grade 2 in 1935. The cervical lesion healed and she was well until December, 1941, when she returned complaining of cough and loss of weight. She died in January, 1942, and autopsy showed metastatic carcinoma of the upper lobe of the left lung, healed carcinoma of the cervix and metastatic carcinoma of the liver. The second case was in a woman of sixty-six treated in 1938 for a Grade 2 carcinoma of the cervix and in 1939 for a papillary growth in the anterior wall of the vagina. She remained well until October, 1941, when she returned complaining of a burning sensation in the region of the left scapula, precordial distress and pain in the left flank. She died with metastatic carcinoma of the liver, lung and brain.

Cervical carcinomas have always been thought to spread only through the lymphatics. But these cases show that when the patients survive for longer periods of time as they do with this treatment blood metastases occur to the bone, liver, lungs and brain and the more life is prolonged the more common such metastases will become.

Two hundred thirty-four patients with car-

cinoma of the uterus were admitted to the Gynecologic Service of the Mercy Hospital Institute from May, 1933, to January 1, 1938. Of these 72 were primary carcinomas of the cervix not previously treated. Of the 3 cases in clinical Group I all are alive after five years, of the 16 cases in Group II 50 per cent are alive, of the 32 cases of Group III, 43 per cent and of the 21 cases of Group IV, 9.5 per cent. The five year survival rate for the whole group is 37.5 per cent. These cases show that 75 per cent of cases of Groups I and II can be arrested but that the majority of cases do not come for treatment until they have reached Groups III and IV in which only 25 per cent can be arrested.—*Audrey G. Morgan.*

GARCIA, MANUEL. Tissue dosage in the control of carcinoma of the cervix. *Radiology*, May, 1943, *40*, 463–473.

The author analyzes 191 cases of primary carcinoma of the cervix treated at the Charity Hospital, New Orleans, from April 1, 1938, to August 31, 1939. The absolute three year survival rate for the group was 37.7 per cent. Eight cases are eliminated because the data recorded were insufficient for calculation or because they were not treated. Two patients who died from complications of radium therapy were excluded. Among the 72 survivors 5 had recurrences before the end of the third year.

A combination of roentgen and radium therapy was used whenever possible. The question of specifying the dose in a common unit for all the qualities of radiation used so that the amounts used can be added is discussed. While both roentgen-ray and radium doses can be calculated in roentgens the doses in r cannot simply be added. Quimby has shown that the biologic effect is not a constant function of the amount of radiation when the average or effective wave length is changed. The best method is conversion of the computed doses to the equivalent amounts in one of the qualities used. Graphs are given showing the method of conversion and tables showing the doses attained.

It seems possible to give doses of radiation which are well tolerated and which give three year recovery in about three-fourths of Stage I and II cases and in nearly half of Stage III cases.—*Audrey G. Morgan.*

TRAUT, HERBERT F. Uses and abuses of radiation therapy in obstetrics and gynecology. *Am. J. Obst. & Gynec.*, 1942, *44*, 638–647.

In this paper the author assesses the value of radiation therapy in obstetrics and gynecology from the more general point of view. He considers the subject from the standpoint of the age period in the life of a woman.

Pre- and Postpuberal Period (first two decades). (1) The use of irradiation should be confined to the treatment of the rare instances of malignant disease. (2) An active stand in restraining the use of the so-called "stimulating" or "temporary castrating" dose of radium or roentgen rays should be taken.

The Reproductive Period (third and fourth decades). (1) The treatment of carcinoma of the cervix constitutes the greatest use which can be made of irradiation during the reproductive period of life. (2) Very rarely may irradiation be indicated in a case of endometriosis. (3) The use of castration in treatment of cancer of the breast in women of reproductive age may be warranted. (4) Seldom will irradiation be the treatment of choice in a myoma occurring in this age period.

Pre- and Postmenopausal Period (fifth and sixth decades).

(1) Hyperplasia of the endometrium, endometriosis, the submucous myoma, and adenomyoma all may be treated efficiently by means of irradiation. Here, roentgen rays may be preferable to radium when there is danger of infection in the uterus or adnexal organs. Intracavitary radium in 1,400 to 1,600 mg-hr. doses is quicker and in many instances satisfactory.

(2) In treatment of pruritus of the vulva and anal region, roentgen treatment is not wise except perhaps in such small amounts as 75 r for five treatments. Irradiation may be followed by abnormal skin changes closely analogous to kraurosis.

The Use of Radium and Roentgen Rays in the Treatment of Malignant Neoplasms.

(1) Carcinoma of the vulva should never be treated by any form of radiation.

(2) In the treatment of carcinoma of the cervix, one of the most important recent improvements has been the application of deep roentgen therapy directly to the cervix and parametrium by means of the intravaginal cone.

(3) The lack of constant and competent gynecologic observation of women in the cancer age constitutes a terrible omission because such observation is the only way in which the *early stages* of the disease may be detected.

Radiation Therapy in the Aged with Advanced Stages of Malignancy.

(1) Sometimes marked regression of the malignant process results from treatment or pain is greatly relieved.

(2) Frequently, instead of giving comfort and prolonged life, irradiation produces nausea, vomiting, and added pain. Roentgen treatment of desperately ill or very aged patients with extensive metastatic disease is to be deplored with very few exceptions. It is a great mistake to conclude that all patients with inoperable or recurrent cancer should receive radiation therapy for often more harm than good is done.—*Mary Frances Vastine.*

SCHEFFEY, LEWIS C. Malignancy subsequent to irradiation of the uterus for benign conditions. *Am. J. Obst. & Gynec.*, Dec., 1942, *44*, 925–951.

Of 481 patients with cervical carcinoma seen on the gynecologic ward service at Jefferson Medical College Hospital between 1921 and 1942, there were 7 who had received radiation therapy for an apparently benign condition, from two to eleven years prior to the diagnosis of malignancy. Conclusions relative to the cervix group are:

(1) All the patients were multiparas, ranging in age from forty-two to sixty-one. Treatment was for fibromyoma in 5 cases and for functional bleeding in 2 cases.

(2) Squamous carcinoma developed in every patient.

(3) There is no evidence to support a statement regarding the presence or absence of malignancy at the time of the initial irradiation, with the possible exception of the 1 patient in whom biopsy was performed. Errors of omission may be charged to the treatment of 6 of these patients when they were first seen. Interest was primarily centered on the fundal lesion.

(4) There is no substantial evidence to show that radiation therapy of the uterine fundus either retarded or accelerated the development of the cervical malignancy in these patients.

(5) *Whenever diagnostic curettage is indicated, the procedure might well be accompanied with cervical biopsy as a matter of record, but especially if any abnormality is apparent.*

Of 124 patients with fundal carcinoma seen during the same period, there were 13 who had received radiation therapy for supposedly benign lesions, from two to twenty-three years prior to the frank diagnosis of malignancy. Conclusions relative to the fundal group are:

(1) Nine of the patients were multiparas, 4 were nulliparas; the ages ranged from forty-three to seventy-one. Four patients were treated for fibromyomas, 5 for postmenopausal functional bleeding and 2 for typical hyperplasia.

(2) Adenocarcinoma was the diagnosis in 12 patients and myosarcoma in one.

(3) Carcinoma was perhaps present in 6 patients at the time of the initial irradiation therapy. In 4 patients in whom malignancy was discovered from ten to twelve years later, no errors of omission seem to have played a part.

(4) In only 2 instances is there factual evidence to indicate that radium may have retarded a malignant growth.

(5) *Radiation therapy should always be preceded or accompanied by diagnostic curettage.* Fibromyomas should never be regarded as the sole cause of postmenopausal bleeding until an accompanying adenocarcinoma of the endometrium has been ruled out. So-called functional or climacteric bleeding should be viewed with similar suspicion.—*Mary Frances Vastine.*

ALDRIDGE, ALBERT H. Intestinal injuries resulting from irradiation treatment of uterine carcinoma. *Am. J. Obst. & Gynec.*, Nov., 1942, *44*, 833–857.

In this paper, the author's experience in the diagnosis and treatment of intestinal irradiation injuries at the Woman's Hospital for a five year period is recorded. Approximately 1 out of every 6 patients, or 16.9 per cent, treated for uterine carcinoma developed some degree of proved injury to the intestine. The total number of patients with intestinal irradiation injuries amounted to 38. Twenty-nine of these 38 cases had intestinal strictures.

Intestinal complications resulting from irradiation injuries include three characteristic types: (1) an acute localized proctitis or proctosigmoiditis; (2) ulceration of the mucosa and wall of the intestine; (3) formation of varying amounts of perirectal fibrous tissue.

Acute localized proctitis or proctosigmoiditis. Characteristic intestinal symptoms appear during the course of treatment or soon after its completion. There is a localized inflammatory process involving the anterior wall of the rectum and distal end of the sigmoid at about the level of the cervix. The symptoms disappear soon after termination of radiation therapy leaving no evidence of damage to the mucosa or wall of the intestine.

Ulceration. The symptoms of ulceration make

their appearance at any time from soon after termination of radiation therapy to within several weeks or months later. They tend to heal slowly and when healing is complete the mucosa is pale and atrophic in appearance.

Perirectal involvement. In this type of reaction, a diffuse mass of fibrous tissue involving all the pelvic structures below the uterocervical junction and extending upward and backward to the second or third sacral vertebra is formed. This manifestation of irradiation reaction is difficult to differentiate from the so-called "frozen pelvis." Intestinal obstruction is apt to be the final result.

It is frequently difficult to differentiate between intestinal obstruction caused by tissue reaction to irradiation injury and conditions resulting from malignancy.

In conclusion, it is noted that sufficient irradiation to effect a cure of uterine carcinoma cannot be applied without some damage to the intestinal tracts of a considerable percentage of patients treated. The development of intestinal symptoms and especially those of intestinal obstruction at any time from a few weeks to several years after irradiation for uterine carcinoma should always suggest the possibility of a post-irradiation intestinal injury.—*Mary Frances Vastine.*

Ward, George Gray. The diagnosis and treatment of carcinoma of the corpus uteri based on experiences at the Woman's Hospital. *Am. J. Obst. & Gynec.*, 1942, *44*, 303–309.

The series includes 192 cases of carcinoma of the corpus uteri observed during the period from 1919 to 1941 inclusive. Ewing's classification was employed in the histopathological grading of these carcinomas:

Grade 1. Superficial papillary adenoma malignum.
Grade 2. Adenoma malignum.
Grade 3. Adenocarcinoma.
Grade 4. Diffuse anaplastic carcinoma.

Grade 1 is of low malignancy while Grade 4 is very malignant, anaplastic, and consequently radiosensitive.

Symptoms. Metrorrhagia, spotting or a blood-tinged watery malodorous discharge was the predominant symptom occurring in 95 per cent of the cases.

Diagnosis. Curettage is necessary as microscopic examination of the growth is essential for the diagnosis. (It was noted that the passage of the uterine sound produced profuse bleeding which was absent in normal cases.)

Prognosis. Grades 1 and 2 give a high percentage of cures if they are treated in the early stages. The prognosis is more grave in Grades 3 and 4. The time lost before the patient seeks relief for the irregular bleeding or discharge has been estimated to average seven to eleven months.

Treatment. (1) Surgery plus irradiation is the treatment of choice. Intracavitary irradiation at the same time the preliminary diagnostic curettage is performed should be done. The author inserts two 50 mg. capsules of radium in tandem formation with 1 mm. of platinum and rubber screening in the fundus. If the diagnosis is "malignant," a dosage of 2,400 to 4,000 mg-hr. is given. The operation is done four to six weeks subsequent to this. (2) 53.6 per cent of this series of cases were poor operative risks so irradiation alone was done in these cases. Repeated irradiation is advisable, a dosage of 2,400 to 4,000 mg-hr. being given at four week intervals. Whenever feasible, deep roentgen therapy should precede or follow the intracavitary treatment. (3) In the cases in which both the cervix and corpus uteri are involved, treatment should be that of carcinoma of the cervix.

Results. (1) Operable cases of corpus carcinoma treated by irradiation, surgery and roentgen rays should average 70 per cent five year survival. (2) Inoperable cases with irradiation alone should show an average five year survival of 35 per cent.

(In view of the more frequent association of carcinoma with fibromyoma—35 to 40 per cent—it is best to remove the entire uterus in all such cases. The removed specimen should always be opened at the time of operation so that a carcinoma of the corpus will not be overlooked.)—*Mary Frances Vastine.*

Judd, Edward S., Jr., and Priestley, James T. Treatment of gastric ulcer. *Surg., Gynec. & Obst.*, July, 1943, *77*, 21–25.

There are many points brought out in this article which should prove of interest to the roentgenologist, viz.:

1. Approximately 7 per cent of patients who undergo resection for malignant growths of the stomach are less than forty years of age.

2. Eighty per cent of patients who have gastric resection for malignant tumors and who previously received medical treatment for

presumed benign ulcer experienced temporary relief from this form of therapy.

3. Approximately 1 of 5 patients who has gastric resection performed for cancer has normal or elevated values for gastric acids.

4. Although 9 out of 10 benign gastric ulcers which are removed surgically are smaller than a quarter, it has been noted that approximately one-fifth of the carcinomatous lesions removed from the stomach have an area of ulceration 4 cm. or less in diameter.

5. Over a period of many years at the Mayo Clinic the diagnosis of gastric ulcer has been made by the roentgenologist in approximately 10 per cent of cases in which resection has been performed for actual carcinoma.

6. The only method of certain differential diagnosis between benign and malignant gastric ulcer is prolonged observation under medical treatment. Such observation should be carried on (intermittently following the first several months) for a period of at least several years.—*Mary Frances Vastine.*

Ellis, F., Wilson, C. W., Dobbie, J. L., Grimmett, L. G., and Green, Anthony. Beam direction in radiotherapy; symposium. *Brit. J. Radiol.*, Feb., 1943, *16*, 31–43.

As the volume of tissue irradiated decreases the dose tolerated by that volume increases. In order to reduce the constitutional effects and increase the local dose, dosage must be very accurate. This can be accomplished by a number of devices for accurate beam directioning. A number of these are described and illustrated, showing their method of application. The entrant and emergent ray method is described which utilizes a surface body mark on each side of the tumor for localizing purposes. The directional caliper used in this method is illustrated. If the depth of a tumor below a given skin point is known and the point of entry of the beam is known, the required direction of the beam and the depth of the tumor can be determined by completing a parallelogram. A pocket instrument for such parallelogram determination is described and illustrated. The arc and pointer method for localizing foreign bodies was worked out in World War I. The same principle can be applied to beam directioning for tumors and the method is described and two arc beam directors are illustrated. An ideal method of irradiating a block of tissue uniformly is to place the tissue inside a three- four- or five-sided figure and compound a series of isodose charts to secure uniformity. Such shapes for the marking out of fields are shown.

If pressure is exercised on a roentgen-ray applicator so that it is squeezed closer to the tumor, it is obvious that the dosage to the tumor is increased with the same skin dose. A method for accurately directing and measuring such compression is shown, as is also a method of keeping the patient motionless during treatment. A longitudinal steel bar is screwed to the side of the treatment couch. A master clamp moves along this bar and can hold various types of body supports for holding the patient in different positions according to the type of application of the rays.—*Audrey G. Morgan.*

Quick, R. S., and Roberts, J. E. Short distance x-ray therapy with standard apparatus; physical factors. *Brit. J. Radiol.*, March, 1943, *16*, 82–85.

The Siemens Chaoul tube and the Philips Metalix tube have been designed for the treatment at low voltages and short focus-skin distances of lesions on the skin or in body cavities. These treatments can be carried out, however, with simple modifications of the standard apparatus. There are lesions, however, in which the lethal dose should reach depths of 1 to 2 cm. and the overlying tissues should not be too heavily irradiated and the dose rate at greater depths should decrease as rapidly as possible. This type of treatment requires some compromise between the standard "deep" and "contact" techniques. It is called middle distance superficial therapy. The authors describe simple, short distance, small field applicators which they have had made for this purpose which they attach to their 200 kv. therapy tubes. With these tubes the most satisfactory focus-skin distance is 22.5 cm. or 26 cm. when applied to the Metropolitan Vickers continuously evacuated tubes. They give tables and graphs showing the radiation output, quality and depth dose measurements with this type of treatment. Modifications of the middle distance applicators to be used in the treatment of intraoral cancers are described and illustrated.—*Audrey G. Morgan.*

MISCELLANEOUS

Walker, Stella F. Classification and filing in department of roentgenology: standard nomenclature. *Radiology*, June, 1943, *40*, 603.

If each hospital adopts its own classification

it is apt to grow and become confused and fail to develop logically. The author therefore suggests the adoption by hospitals of the Standard Nomenclature of Disease which has developed under careful direction since 1928 and has recently been taken over by the American Medical Association which plans constant revision and periodic publication. It would seem comparatively simple to issue a small pamphlet of terms approved by the Radiological Society for use in connection with the Standard Nomenclature. All refinements of the classification need not be used in every hospital but the use of a uniform classification by different hospitals has obvious advantages.—*Audrey G. Morgan.*

Barnard, Virgil L. A new surgical table top and cassette holder for surgical roentgenographic examinations of the hip. *Radiology*, June, 1943, *40*, 599–602.

One of the hardest tasks in roentgenography is to get a true lateral projection of the head and neck of the femur. A surgical table top and cassette holder designed to accomplish this purpose are described and illustrated. Both anteroposterior and lateral projections of the head and neck of the femur can be obtained without flexing the thigh or moving the patient. There is no danger of contaminating the surgical field.

The table top is made of flawless plywood covered with formica which is a plastic similar to bakelite. It fits the top of the table and its thickness is such as to place the head and neck of the femur for the lateral projection approximately in the center of a 10×12 inch film. An angle of 127° with its apex toward the patient's head is cut in each side of the table top. The cassette is placed in the angle, with its edge resting on the edge of the table.

It is crowded into the patient's side between the iliac crest and the lower ribs, and held in place by a nurse. The tube is quickly placed in position and the exposure made. There is a sliding tray under the table in which the film for the anterolateral projection may be placed and slid under the patient.—*Audrey G. Morgan.*

Lasnitzki, Ilse. The effect of x rays on cells cultivated in vitro. Part II. Recovery factor. *Brit. J. Radiol.*, Feb., 1943, *16*, 61–67.

Cultures were made of material from the choroid and sclerotic of chick embryos nine to eleven days old, subcultures being made every forty-eight hours. They were irradiated with single exposures of 100 r and exposures of 100 r separated by intervals of forty-eight and ninety-six hours, single exposures of 1,000 r and exposures of 1,000 r separated by an interval of ten days. Tables and graphs are given showing the details of the results. The qualitative effects were the same in all cases; the differences were only in degree. There was always a temporary decrease of mitosis followed by recovery. There was a small wave of abnormal mitosis following the fall and a second wave, accompanied by the appearance of degenerate cells, after the recovery. With the dose of 100 r, mitosis never disappeared completely and the ratio of the mitotic phases, prophase, metaphase and telophase, was much less distorted than after the dose of 1,000 r. With the latter dose mitosis, both normal and abnormal, disappears for a short time and with the return of mitosis prophase is in excess of metaphase for some hours. Degenerate cells occur during the period when the ratio between the mitotic phases is disturbed. The first effect of radiation is obviously to disturb mitosis and upset the normal process of division in some of the cells that are undergoing mitosis at the time of irradiation. These cells form the first wave of abnormal mitosis and the number varies with the size of the dose.

Recovery is shown by the reappearance of normal mitosis, the restoration of the normal ratio between the phases and the disappearance of abnormal and degenerate cells. This occurred forty-eight hours after a dose of 100 r and ten days after a dose of 1,000 r. If a second dose the same as the first was given as soon as the cells showed apparent recovery, the effect of the second dose was greater than that of the first. If the interval between the doses was prolonged the quantitative effects of the first and second doses approximated each other.—*Audrey G. Morgan.*

Medical Research Council. Medical uses of radium. *Brit. J. Radiol.*, 1943, *16*, 54–58.

This is a summary of reports from experimental research centers for 1941. The Radiotherapeutic Research Unit at Hammersmith Hospital, London, reports continued work on the 2 million volt generator being constructed there. Experiments have been carried out on the electrical resistance of various bakelite-graphite mixtures with a view to correlating the electrical properties with composition. A new optical-mechanical method of working out a three-dimensional dosage system is being experimented with.

At the Royal Cancer Hospital, London, experimental work has been carried out, using a dose finder, on the spatial distribution of radiation around groups of radium needles and other sources. The technique of manufacturing large numbers of condenser ionization chambers bath of high and low sensitivity, has been developed. A wax sectional model of the human body has been constructed in such a way that measurements of dose within it can be made at a large number of points. Integral doses can easily be estimated from the results. With the condenser chambers and the model, information has been obtained in regard to the true energy absorption per unit surface dose over a wide range of qualities of radiation.

The Strangeways Research Laboratory at Cambridge has made a quantitative histological analysis of human biopsy material. The histopathology of irradiated sarcomas in the dog has also been studied as well as the histogenesis of skin tumors induced by benzpyrene in mice. Studies have been made of the mechanism of chemical carcinogenesis, the effect of irradiation on growth and differentiation *in vitro*, the effect of iraadiation on the size of nuclei and the inactivation of animal viruses by irradiation.

A number of new experimental methods have been developed at the Department of Medicine, University of Cambridge, dealing largely with ultraviolet radiation.

The Mount Vernon Hospital, Northwood, reports on the biological effect of ionizing radiations, a technique for administering known doses of alpha radiation and the production of various injuries and inhibition of mitosis in bean roots by alpha radiation. Cell degeneration following relatively large doses of roentgen or radium radiation has been found to be due to the breakdown of cells in the delayed mitosis following irradiation.

The Barnato Joel Laboratories of the Middlesex Hospital, London, report on experiments showing the biological effects of radon irradiation of rabbits' ovaries.—*Audrey G. Morgan.*

Hawley Sydney J. Increased filtration for diagnostic purposes. *Radiology*, April, 1943, *40*, 387–390.

It is customary in roentgenographic work to make a roentgenogram of high contrast by using low voltage and little or no filtration. While this gives a brilliant roentgenogram it may sacrifice the sharpness of outline necessary for diagnostic purposes. The relative densities of many parts of the body are similar and disease may cause little change in density. Therefore, the increased sharpness of outline produced by increasing the filtration, say from 1 to 3 mm. aluminum, with a corresponding increase in voltage or exposure time, makes better diagnosis possible by producing increased sharpness of shadows and increasing the range of values that can be obtained. Roentgenograms are given illustrating the difference in roentgenograms taken by the usual method and with increased filtration.

The authors claim no originality for the method. The idea was suggested by Allen and Calder in the *British Journal of Radiology* and they present it to call it to the attention of American readers.—*Audrey G. Morgan.*

Atlee, Z. J., and Trout, E. D. A study of roentgen-ray distribution at 60–140 Kv.P. *Radiology*, April, 1943, *40*, 375–386.

Study of the distribution of radiation around the roentgen tube is particularly important in superficial therapy where large fields are used at a short focal distance. The authors studied the effect of several factors on distribution at a range of voltage from 60–140 kv. (peak) with most of the work carried on at 100 kv. (peak). A tube current of 5 ma. was used in all measurements. A Victoreen condenser r-meter was used and all measurements made at the same focal chamber distance. The chamber was first centered directly below the focal spot. The intensity at this point was taken as 100 per cent and is referred to as the "central beam." The intensity at other points was plotted in percentages of this central beam. At 60 and 100 kv. (peak) studies of filtered radiation were made using 2 mm. aluminum. At 140 kv. (peak) the filtered studies were made with a filtration of 0.25 mm. copper and 1.0 mm. aluminum.

Graphs are given showing the details of the results with different types of tubes. Many of the methods used in superficial therapy were established around the Universal type Coolidge tube. So distribution around this type of tube was studied before that around the more modern types such as the oil-immersed superficial and medium therapy tube and the 20 target oil-immersed roentgenographic type tube. Experimental types were also constructed and studied including a 30 target oil-immersed tube, a 45 target oil-immersed tube, a curved target oil-immersed tube and a convex target

oil-immersed tube. The convex target tube gave the best results in treatment. The tubes used for treatment should not be used for roentgenography because of the changes in the pattern of field intensity due to roughening of the target. The target angle of a tube used for superficial therapy should be at least 30°. Field distribution can be improved by using a curved or even a convex target face for a tube that is used only for superficial therapy.—*Audrey G. Morgan.*

Symposium. Protection against radiations in medical and industrial practice. *Brit. J. Radiol.*, Jan., 1943, *16*, 1–7.

After the induction of Prof. Mayneord as President of the British Institute of Radiology and the Industrial Radiology Group of the Institute of Physics he introduced the subject of protection against radiation and said that the normal, healthy human being can tolerate without injury about one international roentgen per week of roentgen or gamma rays. This rate is sometimes known as the tolerance rate and quoted as 10^{-5} r/sec. He also called attention to the growing use of radiation in industry and the necessity of protective measures there. He then introduced Ralston Paterson who spoke on the Effects of Radiation on Workers and S. Russ who discussed Accepted Standards in Radiological Protection.

Paterson described the early skin changes in radium workers. They appear in the form of erythema and glossiness of the skin, most marked around the nails. This comes from the handling of radium or radon. Warts may appear on the backs of the hands and undergo cancerous degeneration, though this usually occurs as a result of a single exposure to a large dose rather than to long-continued slight over-exposure. If exposure is continued the results may be fatal.

The effects on the blood of general exposure are manifested first on the white, rather than the red cells. There is an absolute and progressive leukopenia, the decrease being in the neutrophils. Some individuals who are particularly radiosensitive show an absolute lymphocytosis with absolute neutropenia as the first sign of over-exposure. White cell counts should be made at least monthly, but a monthly count on individuals for an adequate period would require considerable time. Taking the average white cell count of a group of workers is suggested. If the average count of a group of 10 workers registers below 5,000 it proves that the working conditions are unsatisfactory.

Workers in medical radiology had to pay by bitter experience for their early lack of knowledge of the effects of the rays. Industrial workers should be protected by the knowledge acquired and rules for protection should be enforced by law.

Russ, speaking on accepted standards in radiological protection, called attention to the fact that it has been found that many young people engaged in radium luminizing show a lymphocytosis. It is questionable whether this is due to radon in the circulation or to gamma radiation from the radium which the workers handle. It would be useful to know whether the effect is caused by alpha or gamma radiation as the protective measures would be different in the two cases. Tests have been made in the last six months on the radon content of the expired air of workers. Generally radium workshops and luminizing rooms have a radon content of 10^{-11} curie per liter. People who work in such rooms show radon in the expired air which may be due to living in a radon atmosphere or to actual ingestion of radium. If it is due to radon alone a positive test on Friday will have become negative by Monday but if there is radium in the body it is still positive. It is believed the working atmosphere should not be above 10^{-10} curie per liter. It is harder to fix a safety figure for radium. Even normal human tissues contain some radium. If after a week-end test a person who has been handling radium for a long time shows a radon content in the expired air of 10^{-11} curie per liter it is almost certain that some radium has been ingested and the work should be discontinued.

If these figures survive critical discussion he hopes they may be incorporated in the sixth edition of the recommendations of the Radium Protection Committee which is forthcoming.—*Audrey G. Morgan.*

Binks, W. Protection in industrial radiology. *Brit. J. Radiol.*, Feb., 1943, *16*, 49–53.

The current recommendations of the British X-Ray and Radium Protection Committee say that roentgen-ray workers can stand about 1 international roentgen of either kind of radiation in a week. They should not be exposed to more than that in industry any more than in medicine. Workers with the roentgen-ray apparatus

used for crystal analysis sometimes expose themselves to the direct beam for a short time with resultant severe injuries to the fingers. It should be possible to avoid such injuries by putting up a guard plate to protect the hands. The manufacturers of roentgen tubes try to produce self-protected tubes by intercepting the unwanted direct radiation by surrounding the tube as completely as possible with protective material of adequate lead equivalent. Graphs and tables are given showing roentgen-ray outputs for different voltages, the transmission through lead of roentgen rays excited at different voltages from 50 to 200 kv., and the thickness of lead required to reduce the direct roentgen-ray beam to the dose which can be tolerated without injury.

To protect the worker from scattered radiation he should be stationed behind a protective wall with an adequate lead equivalent, not less than 2 mm. for voltages up to 200 kv., or should stand in a lead-protected pulpit. The operator should stand as far as possible from the roentgen tube or radium source and persons not engaged directly in roentgen or radium work should not be allowed to enter the danger zone. —*Audrey G. Morgan.*

SPIERS, F. W. Materials for depth dose measurement. *Brit. J. Radiol.*, March, 1943, *16*, 90–97.

The factors that determine the suitability of different materials for depth dose measurement are electron density and the effective atomic number. These were determined for a number of materials including rice, sugar, borax, sodium bicarbonate, boric acid, powder mixtures and waxes and pressdwoods. Graphs and the mathematical formulae used in the determinations are given. Water was found to be the most satisfactory medium for depth dose determinations in spite of the difficulty of using a liquid medium. Its electron density and atomic weight are practically the same as those of the soft tissues. Neither pressdwoods or waxes seem to be safe for use at wave lengths greater than 0.2 Å, particularly in low voltage therapy. If any material is to be used for the measurement of depth doses it should be compared with some standard, such as water, before using.—*Audrey G. Morgan.*

REINHARD, M. C., and GOLTZ, H. L. Quality, area, and distance relationship for D_5, D_{10}, and D_{15}. I. *Radiology*, March, 1943, *40*, 283–292.

Up to 1940 the New York State Institute for the Study of Malignant Diseases had worked with voltages not higher than 200,000 but at that time a million volt and a 400,000 volt roentgen generator were added. Knowledge of the depth measurements for these generators was limited. The authors therefore decided to assemble all the depth measurements made at this Institute for a number of years, together with the published results of other authors, in order to establish a relationship between depth intensity and size of field, quality and distance, so that depth intensities for one set of these conditions could be converted simply to another set. Ratio curves for distances of 5, 10 and 15 cm. from the surface are reproduced, providing a means for establishing depth intensity curves for any set of conditions used in roentgen treatment with from 200 to 1,000 kv. A depth dose table is given covering a range in quality from 0.9 to 9.0 mm. copper half-value layer, areas from 25 to 400 sq. cm. and distances from 50 to 100 cm.—*Audrey G. Morgan.*

REINHARD, M. C., and GOLTZ, H. L. Quality, area, and distance relationship for D_5, D_{10}, and D_{15}. II. *Radiology*, March, 1943, *40*, 293–296.

In a preceding article the authors gave tables and curves showing the relationship between depth intensities and the three factors of quality of radiation, size of field and focal skin distance at depths of 5, 10 and 15 cm. from the surface. They demonstrated a method of transposing a known depth dose from one set of these conditions to any other within the range 0.9 to 9.0 mm. copper half-value layer, 25 to 400 sq. cm. area treated, and 50 to 100 cm. focal skin distance. This paper presents an extension of this work. The depth was extended to 20 cm. and tables and curves are given from which it is possible to calculate depth doses within the range of 2 to 12 mm. aluminum half-value layers, 20 to 200 sq. cm. area and 20 to 60 cm. distance.—*Audrey G. Morgan.*